MEDICAL PROCEEDINGS

MEDIESE BYDRAES

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EDITORIAL · REDAKSIONEEL

MEDICAL HAZARDS OF BOXING

In this issue we publish an important survey by Mr. David A. Muskat on *The Problem of Death in Boxers from Cerebral Injury.* Mr. Muskat is particularly well qualified to deal with this subject, as his experience is by no means confined to that of the onlooker (whether in the capacity of a medical practitioner or a spectator). He has had intimate experience inside the ring, having been the Witwatersrand University Featherweight champion for 3 years running.

It is clear from Mr. Muskat's review that there is a disturbing and increasing incidence of deaths during and following boxing bouts. The position is not peculiar to South Africa, as the same tendency has been observed overseas. Equally disturbing, and less commonly referred to, are the hazards of increased morbidity, which involve not only the performers in the ring, but also the much vaster army of sparring partners, who depend on this occupation for their livelihood, in whole or in part.

Although amateur boxing had not been associated with the same incidence of the fatalities which mark professional boxing, serious and even fatal results have been known to occur.

The proponents of the view that boxing is justified on the grounds that it encourages 'the noble art of self-defence' could hardly rest their case on a weaker foundation. The art of self-defence could, with greater profit, be taught with considerably less risk to life and limb by practitioners of such pastimes as ju-jutsu; and there is much to be said for encouraging this knowledge, even amongst the school-going population (both boys and girls).

DIE MEDIESE GEVARE VAN BOKS

In hierdie uitgawe publiseer ons 'n belangrike oorsig van *The Problem of Death in Boxers from Cerebral Injury* deur mnr. David A. Muskat. Mnr. Muskat is by uitstek bevoegd om hierdie onderwerp te bespreek, want sy ondervinding is geensins beperk tot dié van toeskouer (òf in die hoedanigheid van mediese praktisyn òf as gewone toeskouer) nie. Hy het intieme ondervinding van wat binne in die kryt self plaasvind, want gedurende 3 agtereenvolgende jare was hy veergewigkampioen van die Universiteit van die Witwatersrand.

Uit mnr. Muskat se oorsig blyk duidelik dat dat daar 'n onrusbarende toename is in die aantal sterfgevalle wat tydens en ná boksgevegte plaasvind. Dié posisie is nie eie aan Suid-Afrika nie, want dieselfde neiging is ook in die buiteland waargeneem. Ewe verontrustend, maar nie so in die oog lopend nie, is die gevaar van verhoogde morbiditeit nie alleen onder vertoners in die kryt nie, maar ook onder die veel groter leër van skermmaats wat geheel en al of gedeeltelik van hierdie beroep vir hul lewensbestaan afhanklik is.

Hoewel minder noodlottige ongelukke in die amateur- as in die professionele bokskryt voorkom, is ernstige en selfs dodelike gevolge onder amateur-boksers nie onbekend nie.

Die voorstanders van die sienswyse dat boks geregverdig is op grond daarvan dat dit ‚die edele kuns van selfverdediging' aanmoedig, kon hul argument beswaarlik op 'n swakker grondslag gebaseer het. Die kuns van selfverdediging kan met groter voordeel en met aansienlik minder lewens- of beseringsgevaar deur beoefenaars van sulke tydverdrywe soos ju-jutsu gedoseer word; en sterk argumente kan

It is undesirable, however, to impose virtue on a reluctant population by compulsion, and the argument will no doubt be advanced that some regard must be had to the almost instinctive sporting desire of men to engage in combat, even if there is a fatal risk to the participants. On this view, boxing has the same moral standing as such diversions as bull-fighting or cock-fighting.

Mr. Muskat's paper makes clear that boxing has passed from a sport to a vast organized industry, a situation that carries with it many undesirable features. If it is to continue, increasing preventive and protective measures must be taken on behalf of those who risk death or serious morbidity by earning their livelihood in the ring. We should pay serious attention to such statutory provisions controlling boxing, as e.g. in Belgium, where the State is not prepared to leave the matter to voluntary control alone.

The existing rules and regulations in South Africa are very enlightened, but they leave considerable room for improvement, especially in the field of what Mr. Muskat refers to as 'proper, full and unrestricted medical supervision.'

Mr. Muskat has set out fairly fully the minimal conditions under which the industry and the sport should be allowed to continue. His views should receive the serious consideration of those who are determined to preserve this form of lawful assault.

Mr. Muskat's contribution carries an additional feature of interest for the pathologist. He has put forward his own theory of the sequence of events which occurs in the brain stem in the usual knock-out blow. His views on the mechanism whereby a fatal injury is produced may not meet general acceptance, but they are certainly a stimulating presentation which should focus much desired attention on this important subject.

aangevoer word vir die aanmoediging van hierdie kennis selfs onder skoolkinders (sowel seuns as dogters).

Dit is egter onwenslik om deugdelikheid deur dwangmaatreëls op 'n teësinnige bevolking af te dwing, en die argument sal waarskynlik geopper word dat byna instinkmatige sportiewe verlange van mans om aan 'n geveg deel te neem, nie oor die hoof gesien kan word nie—selfs al bring so 'n geveg ook noodlottige gevare vir die deelnemers mee. Wat hierdie sienswyse betref, is daar vir boks dieselfde morele regverdiging as wat daar vir sulke tydverdrywe soos stiergevegte en hanegevegte is.

Mnr. Muskat se referaat bring baie duidelik aan die lig dat boks nie langer 'n blote sportsoort is nie, maar inderdaad 'n reusagtige, georganiseerde bedryf geword het—'n toestand wat talle onwenslike kenmerke met hom meebring. As dit in stand gehou moet word, sal toenemende preventiewe en beskermingsmaatreëls getref moet word namens diegene wat hulle aan die dood of aan ernstige siekte blootstel deur hul brood in die kryt te verdien. Ons behoort ernstige aandag te bestee aan statutêre bepalings in verband met boksbeheer soos dié wat bv. in België afgekondig is, waar die Staat nie bereid is om die aangeleentheid alleen aan vrywillige beheer oor te laat nie.

Die bestaande reëls en regulasies in Suid-Afrika is weliswaar verlig, maar daar is nog aansienlike ruimte vir verbetering, veral op die gebied van ‚behoorlike, volledige en onbeperkte mediese toesig,' soos mnr. Muskat dit noem.

Mnr. Muskat gee ons 'n betreklik volledige uiteensetting van die minimum-voorwaardes waaronder die boksbedryf en die sport toegelaat kan word om voort te bestaan. Sy sienswyse verdien die ernstige oorweging van diegene wat vasberade is om hierdie vorm van gewettigde aanranding in stand te hou.

Mnr. Muskat se bydrae bevat ook 'n uiteensetting wat van belang vir die patoloog behoort te wees. Hy verduidelik naamlik sy eie teorie in verband met die reeks voorvalle wat in die breinstam plaasvind nadat die gewone uitklophou geplant is. Sy sienswyses betreffende die meganisme waardeur die noodlottige besering veroorsaak word, sal miskien nie allerweë aanvaar word nie, maar is nogtans 'n stimulerende verduideliking wat hoogs nodige aandag op hierdie belangrike onderwerp behoort te vestig.

THE PROBLEM OF DEATH IN BOXERS FROM CEREBRAL INJURY

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In recent years and, more particularly, in recent months, deaths during and following boxing bouts, both here and overseas, have shown a disturbingly increasing incidence. With the prevailing intense competition in all spheres of human activity, boxing, too, may be expected to become highly specialized and intensely competitive; perhaps even more so as it is charged with its own peculiar and inherent dangers. Boxing is unique in that the infliction of injury is purposeful and, in professional boxing particularly, almost the sole legitimate objective for promoting the pecuniary advancement of its proponents. To those

'at the top' it can be a most lucrative occupation and Ferlaino[11, 12] goes so far as to regard boxing actually as an industry which, like all other industries, requires health conservation and accident prevention measures. Insurance companies classify it as a 'hazardous occupation' and the problem may well arise whether, as with all industrial organizations, compulsory protection under the Workmen's Compensation Act should not be applied.

In South Africa public opinion has been aroused by the death, within little over a year, of several boxers, both European and non-European, shortly after participation in bouts. Cogent but not irrefutable arguments have again been advanced to abolish professional boxing. The problem whether boxing as such is more dangerous than other forms of sport is still highly controversial, the antagonists arguing that in boxing alone is the intent solely to injure the opponent, often viciously so. It is, however, questionable, from a perusal of the daily press, whether such intent is solely confined to the 'noble' art of boxing.

In 1951 Gonzales,[13] because of two deaths in professional bouts, was stimulated to a survey of similar fatalities in this and other athletic competitions. He found that in 32 years of boxing competitions in New York City (1918–1950), fewer deaths were produced in proportion to the number of participants than occurred in baseball or football (baseball 43, football 22, boxing 21, soccer 2); and far fewer deaths, proportionally, than resulted from *daily* accidents. Critchley[6] found that 207 fatalities associated with boxing were recorded up to 1950. Since then, of course, the number has been considerably augmented.

In a recent review Smith[38] gives detailed figures of known fatalities occurring throughout the world for the period 1918–1957. The recent two European fatalities in South Africa have been included. The list is considered to be sufficiently important to be quoted (with slight modification in tabulation), in detail (Table 1).

Disaster, in boxing, is ever imminent and a study of the literature reveals a disturbing incidence of morbidity and mortality. The writer does not wish to discuss the controversial features of the subject but intends to confine discussion to the cranio-cerebral aspect, as distinguished from other possible causes of serious, even fatal, injuries.

TABLE 1

Year	*Professionals*	*Amateurs*	*U.S.A.*	*Abroad*	*Total*
1918	0	1	1	0	1
1919	0	1	1	0	1
1920	1	0	0	1	1
1921	2	0	1	1	2
1922	0	3	2	1	3
1923	1	0	0	1	1
1924	1	0	0	1	1
1925	6	0	3	3	6
1926	4	0	4	0	4
1927	1	0	1	0	1
1928	3	1	0	4	4
1929	7	2	1	8	9
1930	7	3	10	0	10
1931	3	0	3	0	3
1932		*No deaths*			
1933	5	1	6	0	6
1934	2	0	1	1	2
1935	0	1	1	0	1
1936	5	1	6	0	6
1937	2	0	2	0	2
1938	1	2	2	1	3
1939	1	0	1	0	1
1940	1	3	1	3	4
1941	4	0	2	2	4
1942	4	1	4	1	5
1943	3	0	3	0	3
1944	2	0	2	0	2
1945	3	3	6	0	6
1946	11	3	14	0	14
1947	5	5	6	4	10
1948	5	5	4	6	10
1949	12	7	10	9	19
1950	7	4	4	7	11
1951	3	9	5	7	12
1952	8	9	4	13	17
1953	12	9	8	13	21
1954	3	3	4	2	6
1955	8	1	3	6	9
1956	3	7	3	7	10
1957	3	5	2	6	8
	149	90	131	108	239

PATHOLOGICAL FEATURES

The head injuries of boxing differ in no way from head injuries in general, except insofar as they are legalized forms of coma production by the application of forces to the head. The sport could lend itself, ideally, to a study of the minutiae of forces, propulsion, acceleration, deceleration and other factors conducive to the maximal effect—thus being closely allied to the experimental approach.

It is not appropriate here to enter into a detailed discussion of the causes of concussion[26] (the reader is referred to a previous communication),[27] but one aspect which has not been sufficiently investigated is the role played by derangement of the midbrain and the brain stem. Isolated instances of morbidity and mortality arising from the so-called midbrain syndrome following head injury have appeared in the literature. Duret (1878, 1919) fairly convincingly demonstrated that midbrain injuries could easily be produced experimentally in

animals. Transient signs of such injury are not uncommon after cranio-cerebral trauma, e.g. pupillary and oculomotor abnormalities and states of decerebrate rigidity. Kremer, Russell and Smythe[21] describe 9 cases of severe head injuries subsequently manifesting dysarthria, hypotonia, ataxia, personality changes, disturbances of equilibrium, static tremor, oculo-motor disturbances, parkinsonism and, in some cases, hyperpyrexia. In all these cases ventriculographic studies revealed dilatation of the aqueductus cerebri—accepted evidence of midbrain changes.

McAlpine and Page[25] discuss the case of a 24-year-old boxer who, boxing since the age of 14 years, had been knocked out twice, each occasion being accompanied by a short period of amnesia. He subsequently developed a parkinsonian syndrome with no loss of intellect, possibly due to damage to the substantia nigra and the red nucleus. The likeliest explanation given was trauma from midbrain contusion caused by the knock-out blows. In the discussion that followed Russel Brain[25] recollected a similar syndrome in a boxer he had seen.

Courville,[5] in a detailed and intensive investigation into the effects of closed cranial injuries on the midbrain and the upper pons, classified them under the following headings (where appropriate, the writer has indicated the relative injuries which could occur in boxing):

(*a*) *General Effects*: The 'punch-drunk' state and traumatic encephalopathy in boxers would be the chief manifestations but, as will be indicated later, acute lethal effects may occur without demonstrable pathological changes in the midbrain or the pons.

(*b*) *Effects of Pressure*: These may be produced by local subdural or subarachnoid haemorrhage accumulating around the brain stem and leading to death in a few hours.

(*c*) *Effects of Local Injury*: In an extreme case the midbrain is completely torn across. In less severe cases this mechanism is manifested by haemorrhages and streaky lesions. In all probability, still milder lesions lead to the clinical midbrain syndromes discussed above.

(*d*) *Effects of the Coup-Contrecoup Mechanism*: In all the cases described the occipital region of the patient's projected head struck an immovable object. The haemorrhages tended to be arranged symmetrically in a central antero-posterior line in the tegmental portion of midbrain. The obvious correlation is the boxer's head striking the hard unprotected canvas.

(*e*) *Effects of Sudden Lateral Dislocation of the Brain*: Force is apparently applied to the side of the head in motion, the margin of the tentorium cerebelli serving as a sharp edge against which shearing action is exerted. Although it has been alleged[17] that blows on the side of the head in boxing rarely cause a knock-out, this mechanism (e.g. from a blow on the temple) must obviously come into play.

(*f*) *Effects of Distortion of the Midbrain*: Any supratentorial space-occupying lesion (haemorrhage, oedema, contusion) results in herniation of the ipsilateral uncus into the incisura tentorii (the space of Bichat between the tentorial edge and the midbrain). This wedge-like tongue of brain tissue compresses and distorts the midbrain and the pons.

(*g*) *Combined Effects*: Any or all of the aforementioned effects may be combined in one comprehensive mechanism.

According to Courville the syndromes consequent to the above injuries could be:

1. *An acute failure of vital functions*: a shock-like state, low blood pressure, cold cyanotic skin, slow laboured respiration, feeble rapid pulse, dilated reactionless pupils and death in a few hours.

2. *Subacute failure,* more especially seen in falls on the back of the head, showing essentially a slowly progressive downhill course without any great fluctuation in symptoms, except possibly the delayed development of decerebrate rigidity. In this type there is no 'interval syndrome'.

3. *Marked hyperthermia,* indicating decompensation.

4. *Decerebrate rigidity,* a relatively rare manifestation. Every one of these types has been encountered in fatalities occurring in boxing.

In respect of decerebrate rigidity, Paul[31] made a detailed clinical and pathological investigation of a fatal injury caused by boxing.

An amateur boxer was knocked out by a right cross to the left side of the chin, resulting in classical decerebrate rigidity, a condition in which, experimentally, the lesion is located between the vestibular and the red nuclei.

The autopsy revealed a diffuse subdural haematoma (part of which had been removed surgically), but no *obvious* lesion of the midbrain. The clinical evidence of decerebrate rigidity, however, indicated at least a functional derangement at this site, and Paul put forward the suggestion of a 'strain in the midbrain region' by the forces projected at the time of the blow.

THE FORCES ACTING ON THE BRAIN IN BOXING

Holbourn[16] stressed the factors of injury by rotation, i.e. rotational acceleration as distinguished from linear acceleration. This mechanism was considered more likely to cause local-

ized injury and not diffuse neuronal damage 'wherein particles of brain are pulled apart' by the shear strain. Tissues, particularly of varying densities, can slide across each other by the shearing action engendered by the rotational acceleration, with injurious effects. Strains, therefore, other things being equal, are manifested more markedly at junctions of tissues of different densities.[32]

It cannot be denied that in a perfectly timed 'upper-cut' the forces of rotational acceleration are almost experimentally applied. A boxer can cushion the effect of such a blow by contracting his neck muscles and altering the position of his head, or by 'riding' with the blow and so causing the force to expend itself. In the groggy or dazed state no such protective mechanisms can be brought into play and the boxer virtually becomes the counterpart of Denny-Brown and Russell's experiment with monkeys,[8] except that the acceleration force projected by the opponent's blow cannot be measured. The importance of the speed of acceleration was well demonstrated by Denny-Brown and Russell,[8] who found that acceleration from zero to 28.4 feet per second was necessary before detectable effects on the brain stem could be elicited. In this respect it is well known that the really dangerous 'uppercut' travels only a matter of inches, but at a tremendous acceleration.

When a blow to the head is not of sufficient severity to cause concussion, it may nevertheless affect the cardiac, vasomotor and respiratory functions, giving rise to a condition resembling primary shock. Rowbotham [32] regards such states as occurring in boxers who receive a knock-out blow on the chin. Here, again, the centres affected are those situated in the brain stem region.

Injuries from force can be very varied. The more severe the blow, the grosser and more easily detectable will be the pathological effects, depending on the threshold value of the blow. In this respect, in their graded experiments, Dennys-Brown and Russell observed, in a considerable number of cases, haemorrhages in the upper part of the cervical cord, considered to be due to direct rupture of the vessels from stretching (the more likely explanation) or squashing.

The actual site of the patho-physiological disturbance occurring in boxing is still conjectural. Gurdjian *et al.*[15] conclude, from experimental findings, that severe concussion is due to derangement in brain stem function and is not a 'grade' of cerebral injury but an actual associated finding. Contusions, lacerations, extradural and other haemorrhages, therefore, will be merely concomitant pathological effects. This has been, essentially, the writer's view as well.[27] Contusion and laceration of the brain can occur without unconsciousness, if the brain stem is not involved. According to Gurdjian *et al.*,[15] acceleration and deceleration cause an increase in intracranial pressure which, in turn, cause injurious effects on the brain stem, resulting in the phenomenon known as concussion. Their findings differ from those of Denny-Brown and Russell in that concussion, at least in their experiments, was not always related to the degree of acceleration. They found, too, that cellular changes in the brain stem may occur in mild injuries without loss of consciousness—a fact which may have an important bearing on the pathology of the post-traumatic parkinsonian syndrome. Conversely, according to Nielsen,[29, 30] extensive lesions of the brain stem are not necessarily associated with the comatose state. This would be in accordance with the findings on a boxer (Case No. 4) who was knocked out but recovered later, remaining conscious for a short period despite the presence, as shown *post mortem*, of extensive haemorrhages in the mid brain and the pons.

It must be conceded that the state of disturbed consciousness resulting from a knock-out or knock-down blow is unique. Actual records of clinical examinations during the 'count' cannot be obtained, although Kaplan and Browder [19] attempted such a correlation.

An opportunity was offered by the New York State Boxing Commission to study the effects of blows to the head in the boxing ring. Within 10 minutes of the knock-out, the defeated boxer was fully examined and taken directly to an EEG apparatus. Cinematography was extensively used to correlate the nature of the blows, i.e. the force mechanism, with the effect. The latter demonstrated that blows to the chin or side of the head in the anterior temporal region seemed to produce the greatest number of knock-outs. However, no clinical abnormalities could be detected and headache or dizziness occurred only rarely.

The distinguishing feature of the ordinary knock-out blow, whether less than 10 seconds (i.e. a knock-down) or more, is that 'a vigorous athlete is reduced, in a moment, to an unstrung bulk of flesh, whose weight alone determines its attitude, if indeed, a reactionless man can be described as possessing an attitude at all;'[33] but, on recovery, the 'blackout' can be overcome or compensated to such

an extent that the boxer may, and frequently does, win the bout.

It is this unpredictable feature, this amazing and ostensibly complete and rapid recovery from a state of suspended activity, which sets such a difficult problem in assessing when or whether a fight should be stopped. When recovery is full and complete, it is not reasonable to postulate primary organic damage in the nature of petechial or contusional haemorrhage or even oedema, though these cannot, of course, be completely excluded. It seems more feasible to reason that a sudden cessation of some vital conductivity has occurred. It is well known, e.g., that merely pinching a nerve with blunt forceps without actually causing macroscopic injury, can so interfere with conductivity as to result in complete but temporary paralysis of function of the nerve fibres (neuropraxia). The brain stem, as the name implies, confines in a narrow localized area all the major conducting tracts and fibres, and is the site of important vital centres, so that injury here can have maximal pathophysiological effects.

A recent editorial in the *British Medical Journal*[9] stressed the fact that, though subdural haematomata in boxers are adequately evacuated, the vast majority of the patients still do not recover. This lends support to the writer's contention that these haemorrhages, in such cases, are merely a by-product, so to speak, of the intrinsic damage which the midbrain and the brain stem sustain from a punch to the head or a fall on the floor of the ring. The importance of the space-occupying haematomata must obviously be accepted, but these lesions, unaccompanied by the more important intrinsic damage, should lend themselves to early adequate surgery and respond with complete recovery. Of the 21 deaths quoted by Gonzales,[13] 15 were caused by subdural haematomata. Some of the cases showed scattered petechial haemorrhages and several demonstrated streak-like haemorrhages in the pons. It was considered that rupture of the cortical bridging veins was responsible for most of the haemorrhages, but no adequate reason was given for the failure to recover after surgical evacuation of the clot.

The patho-physiology of the events can perhaps be more fully appreciated by a reference to the peculiar nature of the anatomy of the parts involved.

The tentorium cerebelli is a tough, dense, fibrous sheet dividing the cranial cavity into a large supra-tentorial compartment (containing the bulky cerebral hemispheres) and a smaller, tighter more compact infra-tentorial section. A U-shaped aperture in this membrane allows the passage of the midbrain and the brain stem, connecting the forebrain to the hindbrain. A minimal amount of space intervenes between the crescentic margin and the brain stem, to allow for the passage of cerebrospinal fluid (mainly the interpeduncular cistern) arteries and veins (the space of Bichat). Closing the U anteriorly is the dorsum sellae of the sphenoid bone (the posterior clinoid processes) and the basilar part of the occipital bone. The convex anterior surface of the pons rests on these structures. The midbrain, a segment of the brain stem about 2 cm. long, passes upwards from the pons through the gap in the tentorium and is in direct relationship with the sharp tentorial border and, somewhat more distantly, with the posterior clinoid processes.

It is not generally realized how vascular the brain substance really is. Kaplan[20] vividly demonstrated this fact (Fig. 1). The basic arterial pattern consists primarily of lateral surface and central perforating vessels arising from a main ventral trunk, in this case the basilar artery, some of the branches of which, supplying the midbrain and the pons, run in at right angles to the axis of the brain stem.

Basically, therefore, we have a large bulky mass of soft, inelastic but plastic tissue (the brain) above a rigid membrane (the tentorium) and a smaller mass tightly packed below it. In between is a narrow bridge of brain tissue of considerable intrinsic and extrinsic vascularity. Owing to its greater bulk and what one could call 'sliding capacity,' movements in the upper compartment far exceed those of the lower in range and magnitude. Not only can these movements be in the sagittal or coronal planes but (and this is of considerable importance) a downward displacement of the brain stem may occur from movements transmitted from above, either in acute phases or as a chronic result of space-occupying lesions (trans-tentorial movements or herniations). A sudden expansion of the ventricular system due to a downward thrust wave of cerebrospinal fluid may cause such a displacement, as was shown by Duret long ago.[22] The long surface arteries are put on the stretch and rupture or go into spasm, as may also the short, transverse, intrinsic vessels. The posterior part of the brain stem moves more than the anterior and, as a consequence of irregular shift, rupture of vessels occurs. Johnson and Yates[18] give angiographic confirmation of such ruptured arteries (Figs. 2 and 3).

In lateral displacements of the temporo-occipital lobes, flattening of the brain stem with elongation in the antero-posterior diameter occurs, resulting in stretch of the long penetrating arteries with consequent spasm or rupture (Fig. 4). These writers believe that

the haemorrhages are the result of disturbed blood supply and not due to any associated pathological factor such as a haematoma, and that they are the cause of the fatal outcome.

We can now apply these facts to the mechanism of injury occurring in boxing. The ideal blow for inflicting a knock-out is one which strikes the chin, so transmitting the force through the temporo-mandibular joints to the base of the skull. Such force, especially if transmitted upwards via the upper jaw, may cause rotational effects on the cerebral hemispheres [16] (Fig. 5) or, and the writer feels probably more so, a direct linear continuation through the basisphenoid (Fig. 6). Inertia causes a slight lag of movement of the brain stem (resting against the basisphenoid) away from the advancing base, stretch of vessels and their rupture or spasm. When the force is expended, there is a rebound phenomenon and the moving midbrain and brain stem impinge on the crescentic posterior border of the tentorium (dural reflections can, for practical purposes, be regarded as part of the rigid brain

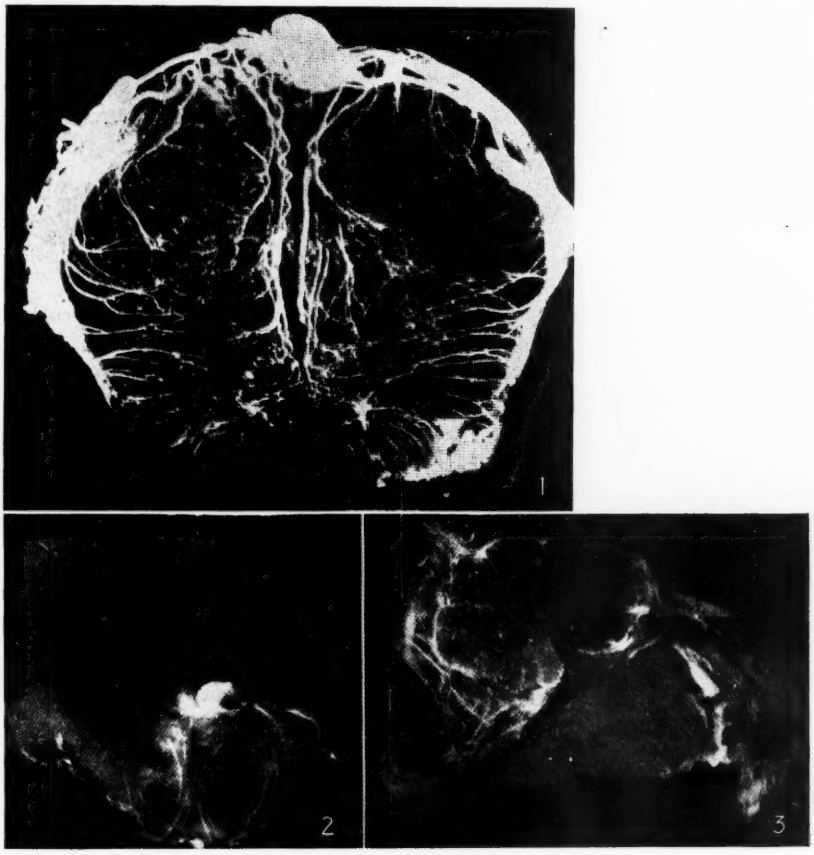


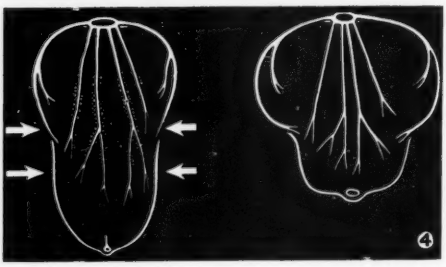
Fig. 1. A cross section of the midbrain showing perforating and surface vessels (From Harry A. Kaplan in *Acta Radiologica*, 1956, **46,** 368.)

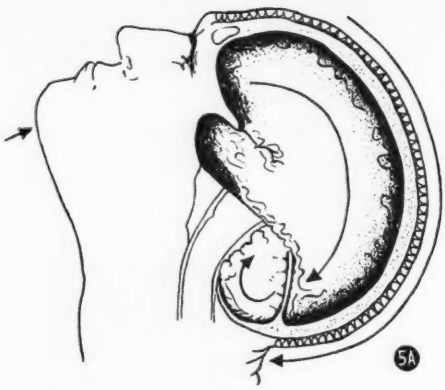
Fig. 2. Radiograph of a transverse slice of the pons from a normal brain. Whilst the arterial injection of silver iodide mixture was being carried out at necropsy, lateral pressure was applied to upper pons and the midbrain. The resulting 'haemorrhages' closely simulate those occurring naturally. (From R. T. Johnson and P. O. Yates in *Acta Radiologica*, 1956, **46,** 255.)

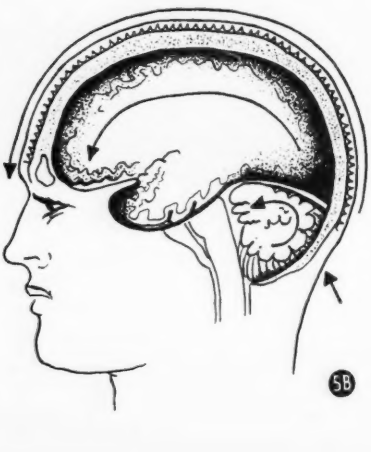
Fig. 3. Photograph of a cleared longtitudinal slice of a normal brain stem. The same procedure was carried out as in *Fig.* 2. In this case, lateral pressure was applied only to the midbrain, and the resultant 'haemorrhages' are confined to this region. (From R. T. Johnson and P. O. Yates in *Acta Radiologica*, 1956, **46,** 255.)

box), the sudden impact again causing injury of both surface and penetrating vessels and even direct injury to the brain stem substance. Pure rotational force, as mentioned before, could set up such stresses and strains at the junctional midbrain area. These could also cause physiological or pathological damage and might be an even more important factor in the production of lesions than is the direct mechanism described.

In Fig. 7 the writer illustrates in detail the intracranial disturbances initiated by a blow to the chin. Fig. 8 depicts the actual blow, as taken during a bout between boxers of international repute. It is this kind of blow, inflicted with the full force available to the assailant, and acting through the mechanism shown in Fig 7, which results in the pathological findings demonstrated in Fig. 10.







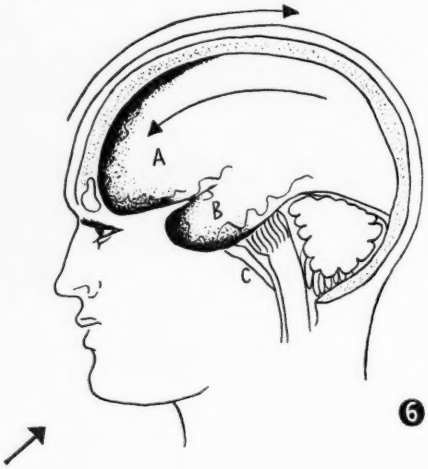
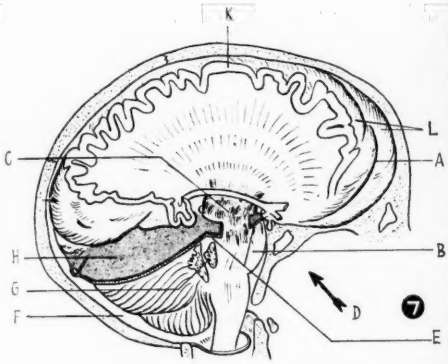


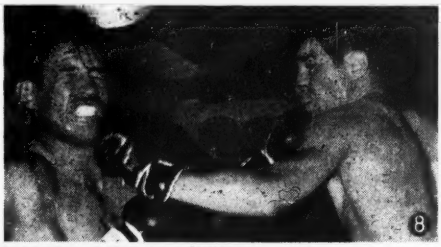
Fig. 4. Diagram illustrating the mechanism by which lateral pressure may, if appropriately applied, cause elongation of the long penetrating arteries and haemorrhages which run back alongside the vessels (From R. T. Johnson and P. O. Yates in *Acta Radiologica*, 1956, **46,** 254.)

Figs. 5*a*, 5*b*. These indicate the conventional conception of the effect of the application of a blow (a) to the chin and (b) to the back of the head (e.g. the head striking the floor) (After Rowbotham).[32] *Fig.* 5a depicts the effect which would classically arise in the groggy boxer with no muscle control. The cerebral and cerebellar hemispheres are presented as moving in rotatory fashion inside the confines of the skull.

Fig. 6. The author has attempted to indicate the major effects of a force applied to the front of the head when the boxer, by tonic muscular contraction, resists rotation of the head. The brain through inertia, lags behind in its movement in relation to the skull, causing *coup* injuries in the anterior (A) and middle (B) cranial fossae. Movements of the pons and midbrain against the base of the skull (C) anteriorly likewise cause varying degrees of stress, strain or injury in this region. (Adapted from Rowbotham).[32]

A fall on the back of the head will reverse the train of events and, as shown by Courville, can be a potent cause of brain stem injury (Fig. 9B). A blow on the side of the head or temple, with lateral displacement of the temporo-occipital lobes, can likewise, though to a considerably lesser degree owing to the resistance of the falx cerebri, cause distortions, strains and vessel injury at the vulnerable midbrain area (Fig. 4). Kaplan and Browder's cinematographic correlation amply demonstrates the importance of such blows despite Jokl's contention [17] to the contrary. Winterstein[36] (as well as Jokl)[17] has sug-





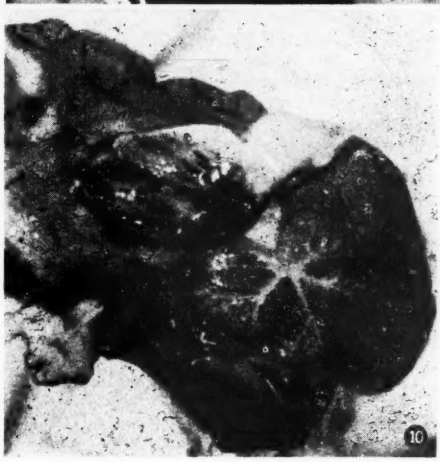


Fig. 7. This shows the author's conception of the sequence of events occurring in the usual knock-out blow as illustrated in the ring-side photograph (*Fig.* 8 reproduced by permission of *The Star*).

The blow transmitted through the jaw does not, in the usual course of events, cause rotatory movements of the skull, but linear movements in the direction of the application of the force.

The skull moves first and the relatively heavy, soft, cerebral hemispheres lag behind. The greater inertia of the cerebral hemispheres results in a greater lag as compared with the smaller cerebellar lobes. The dividing line is at the level of the midbrain (by the tight tentorium cerebelli). Relatively speaking, therefore, the cerebral lobes move forward in the backward moving skull, impinging against the anterior surfaces of the anterior and middle cranial fossae (A). The upper brain stem and pons move against the basisphenoid and the midbrain itself against the clivus (posterior clinoid processes), the anterior border of the tentorial notch (B). The discrepancy between the arcs of movement of cerebrum and cerebellum results in stresses, strains and torsions in the narrow midbrain segment (C).

As discussed in the text, a blow from behind or from the side, or falling on the back of the head all merely alter the direction of linear force, which still causes maximal shear strain at the midbrain level with resultant intrinsic damage or, by impingement on the edge of the tentorium, stretch damage to tracts, internal and surface vessels.

A: *Coup* injury of the frontal lobes.
B: Brain stem.
C: Mid-brain area of stress, strain and torsion.
D: Direction of applied force.
E: Margin of incisura tentorii cut away to show the area of midbrain involved.
F: The brain moves away from the skull.
G: Part of cerebellum cut away.
H: Tentorium cerebelli partially cut away.
K: Portion of cerebral hemisphere removed to show the brain stem tracts.
L: Right and left cerebral hemispheres.

Fig. 8. This graphically illustrates the direction of force impinging on the jaw, the purposeful, tense, re-active muscular contraction of the recipient meeting the blow squarely. One can almost see the jarring on the boxer's expression. By moving his head backward at the time of, or before, the impact (i.e. 'riding the blow') he could have avoided sudden intracranial disturbances and movements and, as a consequence, minimized or avoided concussional effects.

Fig. 10. Photograph of the actual brain damage found, illustrating graphically the site of haemorrhage and maximal damage in the upper brain stem level. It would appear, from the evidence, that the lethal blow in this case was almost identical with that depicted in Fig. 8.

The blow in *Fig.* 8 (according to the mechanism depicted in *Fig.* 7) is, the author contends, the direct cause of the pathology shown in *Fig.* 10.

gested involvement of the medullary centres in a two-phase jerk mechanism (Fig. 9), but this does not adequately explain the major incidence of pathological lesions in the midbrain–brain stem region. Although, of course, medullary lesions could be associated findings, they have been very rare, particularly in fatal boxing injuries.

Thus far the description embraces the grosser evidences of injury. As has been indicated, the short, sharp, concussional force of a knock-out blow can merely result in stress and physiological 'injury' of the vital centres and conducting tracts with the production of unconsciousness. The marked frequency of haemorrhages in these areas (Fig. 10) seems to substantiate the postulated mechanism. We always speak of 'brain' injury when obviously only certain parts of the brain sustain maximally the injurious force. Rowbotham[32] rightly stresses the fact that anatomical units can move in relation to each other so that connecting pathways may easily be bent, stretched or torn during alterations in the shape of the brain. It is contended that even minimal movements of junctional tissues in the midbrain area can result in serious disturbances.

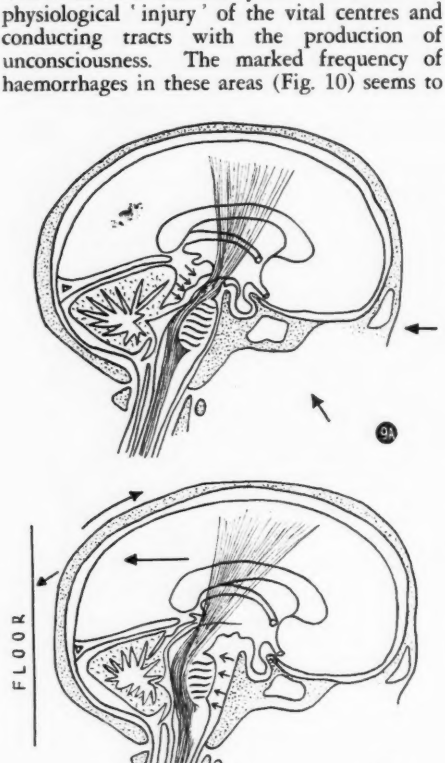


Fig. 9*a* indicates, according to the postulation of Winterstein[36] and Jokl,[17] the movements of the brain stem. A blow to the forehead or the jaw results in relative movement forward of the brain stem (indicated by arrows) causing it to impinge against the basispheno-occipital bone. When the head strikes the floor the movements are reversed (again indicated by the arrows), the brain stem moves away from the basisphenoid and, according to these authors, strikes the margin of the foramen magnum, with involvement of the medullary centres. The writer has tried to indicate in *Fig.* 9*b* more particularly midbrain involvement as against a more predominant medullary involvement suggested by the authors quoted.

Some boxers evince a greater susceptibility to the knock-out blow than others, and are relatively easily rendered unconscious by appropriately placed blows on the jaw. These persons are described as possessing 'glass-jaws'. However, obviously their jaws are not at fault but (by reference to the mechanism already described above) there seems to be, in their cases, an undue sensitivity of the brain stem to dislocating influences transmitted by the blow on the jaw, i.e. an undue vulnerability of the vital centres and connecting fibres. Other fighters can 'take it' and, particularly those of the 'slugger' variety, keep on 'coming for more,' swinging and punching wildly in the hope of winning the bout by a lucky knock-out blow. In doing so they obviously expose themselves to numerous blows transmitting to a relatively stable midbrain system, but still rendering the latter liable to cumulative traumata. These, being repeated innumerably both in sparring and official bouts, eventually result in a chronic midbrain syndrome giving the clinical picture so well described above. This series of events is probably the pathological basis of the state of punch drunkenness. It is also possible that when a boxer is 'softened' for the final knock-out blow, these cumulative midbrain 'concussions' pave the way.

Recent fatal cases 'in the ring' illustrate the pathological sequence postulated above.

Case 1. A young non-European boxer complained of headache the night following his bout. He died 'suddenly' the next morning, while still in bed.

Autopsy revealed a thick layer of subdural haemorrhage covering the upper and outer surfaces of the right cerebral hemisphere. There was some herniation of the right uncus. Section revealed *recent well marked haemorrhages in the midbrain and the pons.*

Case 2. A young non-European boxer received a blow while boxing in the ring and fell back striking his head on the canvas. He was rendered comatose immediately.

On arrival at the hospital his respiratory rate was 6 per minute and both pupils were 'mid-dilated' and fixed. Lumbar puncture revealed a pressure of 300 mm. H_2O.

Death occurred 24 hours later. At autopsy a fair-sized subdural haematoma was found, as well as marked prominence of the left uncus. In addition to contusion of the temporo-occipital lobe, there was an area of *contusion in the left side of the pons.*

Case 3. A young European boxer, after being rendered groggy, was knocked out. Despite surgical treatment, death occurred the next day.

Autopsy showed *a large area of haemorrhage in the midbrain and the pons,* with some involvement of the floor of the fourth ventricle anteriorly. A layer of subdural blood covered the upper surface of the brain. There was *no evidence of uncal herniation.* Small haemorrhages were present in the cerebellum and the left middle cerebellar peduncle, as well as in the occipital lobe and the cerebrum. There were also haemorrhages in the spinal cord at the level of the third cervical segment.

Case 4. A young European boxer was knocked out cleanly. Though he regained consciousness temporarily, eventually he lapsed into deep coma with *decerebrate rigidity.*

Death occurred despite evacuation of a subdural haematoma.

The autopsy revealed *numerous large streaky haemorrhages in the midbrain and the pons,* extending radially into the floor of the fourth ventricle and the upper part of the medulla.

Microscopically there were numerous haemorrhages in the pons. There was prominence of the left uncus.

We know that haemorrhagic lesions of the brain stem accompany supra-tentorial lesions and are believed to be the consequence of supra-tentorial herniation. However, such haemorrhages can occur in the absence of space-occupying lesions and the latter can occur without the haemorrhages. In Case 3, e.g. there was no supra-tentorial coning to account for the haemorrhages. It is the writer's contention that death resulted primarily from the intrinsic nature of the damage localized to the brain stem and that the space-occupying haematomata (in these cases, subdural) were co-effects of the same causative force, aggravating and no doubt contributing to the fatal outcome.

Death occurred despite surgical evacuation and it is contended that these cases, with brain stem damage, are beyond surgical aid. If recovery were to result, a chronic midbrain syndrome would follow. This is probably the position in another case of the boxer Jackie Tiller of Sheffield injured in May 1957, and still apparently in a state of helpless confusion.[34] It would be interesting to see what ventriculography shows in this case, more particularly in connexion with dilatation of the aqueductus cerebri.*

* According to a report in *The Star* on 15 April 1958, he died on 14 April, after having been in coma for 293 days.

The phenomenon of coincidence in our series cannot be invoked, for we have here the severe forms of midbrain and upper pons lesions described by Courville in his series, emphasizing dramatically the mechanism of transmission of forces to the 'weak' spot of the brain. In Case 4 there is virtually the experimental reproduction of the spinal cord haemorrhages described by Denny-Brown and Russell. As mentioned earlier, when a boxer is dazed or groggy, the reflex protective mechanism of muscle splinting is lost, the neck musculature is almost completely relaxed and the head forms an arc of a circle of movement beyond the normal, with excessive effect of forces, transmitted from the jaw or other cranial targets, on the brain stem axis, both torsional and contusional (Fig. 6).

PROPHYLACTIC AND PROTECTIVE MEASURES

Because of the various physical, pathological and physiological factors contributing to danger and death 'in the ring,' and accepting the sport as it is, it becomes imperative, if the game is to survive, to introduce measures which will reduce unnecessary risks to an absolute minimum. The rules and regulations applying in South Africa, although very enlightened, certainly leave considerable room for improvement. For the application of these measures to all phases of the boxing industry it is convenient to discuss the problem under the following headings:

1. The selection of participants.
2. Training methods.
3. The avoidance of excessive punishment in bouts.
4. Avoiding, or reducing, continued exposure to injuries.
5. Periodic checks.
6. The 'materials' of boxing.

Preventive measures do not devolve entirely on those concerned with the promotion and training of fighters (though these have their obligations to their charges).* The most important measure is *proper, full and unrestricted medical supervision.* 'The skilled physician, clothed with authority, is the boxer's best friend.' In America a medical programme for the boxing industry, if not fully accomplished,

*In April 1958 the Belgian Government introduced a Bill stating that 'boxing exhibitions and matches may only be organized under conditions that will protect boxers as far as possible from broken bones and serious external or internal injuries or other wounds.'

Provision is made for the punishment of persons taking part in matches 'contrary to conditions to be laid down by the Health Minister.' (*The Star,* 3 April 1958.)

is at least strongly in the process of being established. It is compulsory to have a physician designated by the Commissioner of the State Athletic Boxing Commission in attendance. In New York State, e.g. a Medical Advisory Board has been appointed, consisting of leading medical men who have themselves been athletes in their day. They would therefore display a deeper interest in and may even have a greater personal experience of the sport. Medical rules and regulations have been promulgated and a better and more thorough medical supervision has been brought into being.

In England in 1929, mainly through the efforts of the late Lord Lonsdale, the British Board of Boxing was established, the principal medical officer acting as one of the medical stewards in an advisory capacity. In 1950 a Medical Subcommittee was formed and the same year witnessed the establishment of the European Boxing Union. More recently a World Boxing Union has been formed.

It must here be emphasized that *amateur boxing* is relatively safe,[35] though serious injuries and fatalities (e.g. Case 1) do occur. 'Many will feel that the benefits from boxing outweigh its dangers.'[9] It is considered that boxing is one form of outlet for psychopathological traits and possibly helps to combat juvenile delinquency. It is, however, unfortunate that the type mostly in need of this form of escape-substitution for the more primitive instincts does not participate more frequently.

1. *The Selection of Participants.* The initial medical examination must not be perfunctory. Obvious disorders, visual and aural defects, locomotor disabilities, epilepsy and kindred disorders preclude participation. Note should be made of intellectual capacity, environmental conditions, possibly of factors concerned with heritage and education and psycho-constitutional factors.[19] These latter loom as important in the possible development of the so-called punch-drunk syndrome; effects could be correlated with causes possibly other than those attributable directly to trauma.

A wise British precautionary measure is to allow only first-series (e.g. champion or near-champion) boxers to fight outside their place of domicile.

2. *The Training Methods.* Boxing should be made safe not only in the actual fight, but also in the training phases. In 'hard' training a boxer can easily absorb a good deal of punishment. It is estimated that the average professional fighter receives 1,200 to 1,500 blows on the head whilst training for an average fight.[19] In the usual 10-round bout, about 1,000 punches are struck to various parts of the body, although no less an authority as Jack Dempsey is quoted as saying that only one blow in a thousand is 'effective' to the head.

A rational approach is made by Egan,[10] who advises a 6-week programme of preparation on the premise that boxing can be adequately taught without the necessity of fighting daily in the gymnasium and thereby subjecting the brain to unnecessary trauma.

The essential features of the programme outlined is the building up of stamina by skipping, shadow-boxing, calisthenics (with emphasis on strengthening the neck and abdominal muscles), sprinting and walking. This may not seem new, but the approach to these methods is different. Correct methods of clinching and avoiding punishment when stunned are important items. Boxing as such is only introduced in the fourth week. In the fifth week 'foul' methods are taught with a view to avoiding them, e.g. 'thumbing' (a cause of detached retina), holding, butting, back-handing and foot-stamping. By the end of the sixth week optimum fitness should have been attained, without having subjected the boxer to the unnecessary punishment to which the usual methods of constant sparring so heavily contribute. Incidentally, one result of such a programme would be a decrease in unnecessary trauma to the sparring partners—usually men who have passed their peak and often themselves becoming candidates for the punch-drunk state.

3. *Avoidance of Excessive Punishment*: This can be met by:

i. An enforced 8-second count after any knock-down, to give the combatant an opportunity to regain his breath, equilibrium, etc. (New York State Boxing Commission).[28] This measure should help to restrain the opponent from 'going in to kill.'

ii. A defenceless boxer should not be allowed to be struck—disregard of this rule by the referee should result in the latter's licence being withdrawn.[22] During 1954, of 1,973 bouts, 360 were stopped before a knock-out could take place.[23] Actually, the spectators themselves protested if the referree did not stop the fight soon enough. Even a few seconds' delay in stopping a fight might lead to a fatal blow being struck.

iii. A fight should be stopped after 3 knock-downs.

iv. At present [19] referees are instructed to declare a technical knock-out whenever a contestant shows inability to protect himself as a result of an unduly prolonged, dazed state. A dazed fighter is obviously an easy target for a knock-out. Item (i) could very well be applied here.

v. If fatigue becomes excessive, the referee may declare a technical knock-out in order to prevent further unnecessary injuries.

vi. The ring-side medical attendant should advise the referee when, in his opinion, the loser is in danger of severe injury.

4. *Avoiding or Reducing Continued Exposure to Injuries.* The following are important:

i. A unit file medical record should be kept for every boxer, to be reviewed and studied from year to year. American football coaches have established a fatality and accident study committee. The same could be done in boxing.

ii. The cessation of all boxing after a knock-out until a full medical examination has been carried out. A 30-day suspension from boxing after such a knock-out has been strongly advised. Even when there is a knock-out with rapid recovery, suspension should be enforced —preferably for a minimal period of 2 months. 'Knock-out prone' subjects should be suspended permanently.

iii. After a knock-out the contestant must be medically examined before contracting to box again. He must obtain a certificate of absolute fitness,[14] otherwise he should be liable to indefinite suspension.

iv. The issue or renewal of a licence must depend on the past medical history, physical fitness, past performance in the ring and, possibly, electro-encephalographic investigation.

v. No boxing should be allowed within 4 weeks of having sustained open wounds of the face 'target area.'

vi. There should be no re-licensing of medically unfit boxers.

vii. Licences should be withdrawn from those showing evidence of cerebral damage.

viii. If a boxer loses more than 4 consecutive fights, enforced examination should be demanded (British Board of Boxing Control).

5. *The Materials of Boxing.* Boxing gloves should be of the optimum size commensurate with practical utility—larger gloves are safer. They should have adequate resiliency (to avoid eye injuries) with fixed padding inside. Mouth guards and head guards should be worn to dampen down the force of the blow.[28]

The construction of the mat is of paramount importance. Sorbo-rubber should be used under the canvas [2]—of a maximum thickness short of impeding the movement of the boxers. Felt is unsatisfactory. The knocked-out boxer, unconscious but still perpendicular, falls with his full weight and effect of gravity, completely toneless, with neck muscles relaxed, striking the floor in a heavy inanimate fashion. This renders the brain liable to the maximal effect of the various forces applied to it (Figs. 5B and 9B). Indeed, many believe that the force of impact in the fall is more conducive to severe and often fatal injury than is the actual blow itself. Kaplan and Browder [19] state that the 5 fatalities autopsied by them over a period of 26 years, all resulted from falls backwards as a result of a blow to the head. Courville,[5] as indicated previously, considers that such falls directly caused midbrain injuries.

6. *Periodic Checks.* The advisability of routine medical examinations has already been stressed. In recent years extensive studies have been made for evidences of electro-encephalographic changes in boxers. To the lay mind an air of mysticism and authority has developed around these studies and a disproportionate importance attributed to the findings, so much so that a normal EEG is regarded as a certificate of fitness to fight and a yardstick of the absence or presence of cerebral damage. Only brief and salient reference to the subject will be made here.

Kaplan and Browder [19] examined a large series (1,043) of professional fighters within 10 minutes of their being knocked out. They came to the conclusion that the EEG was of no contributing value. Continued observations failed to reveal more abnormal records in 'sluggers' as compared with skilled boxers. It was frequently difficult to decide whether changes, when present, were inherent in the individual or secondary to punishment in boxing. The question of changes consequent to possible hypoglycaemia and hyperventilation (in the acute cases) also arose. The role of the EEG in professional boxing has still to be decided, in the opinion of Blonstein and Clarke [3] and, at best is of doubtful value. Critchley [6] found the results contradictory, but in some cases a non-specific type of disordered rhythm was revealed, indicative of cortical atrophy. On the other hand André-Balisaux [1] found unsuspected abnormalities in 68% of cases of isolated subjective post-traumatic syndrome 6 months or more after the occurrence of the injury; but in these cases the persons,

not necessarily boxers, actually had symptoms which, in themselves, would have been detected clinically. Busse and Silverman,[4] although conceding that the value of the EEG is still *sub judice,* consider that, on the whole, those knocked out showed definite disturbances. When focal they were good evidence of brain damage. In their relatively small series of 24 cases, 9 (37.5%) showed an increased incidence of dysrhythmia. In their opinion periodic compulsory examinations should be encouraged and might contribute to the protection of boxers.

To sum up, it is felt that any boxer who exhibits the slightest neurological change, whether mental or physical, should be subjected to electro-encephalographic examination. Positive changes should preclude further participation, even though subsequently the picture may revert to normal. A negative finding is of no significance whatsoever, and does not, by itself, indicate fitness.

A BRIEF NOTE ON 'PUNCH-DRUNKNESS'

This term was coined by Martland [24] in 1929 and has been widely accepted in the literature. More scientific descriptions have been applied, such as post-traumatic encephalopathy and dementia pugilistica, but they do not convey the essential features. The incidence of the condition has apparently been exaggerated. Kaplan and Browder [19] rightly point out that when many physicians are questioned about the extent of their knowledge of the supposedly punch-drunk ex-fighter, they admit they knew nothing of his intellectual capacity during his pre-fight years, his environment, heritage or educational standard.

The pathology of the condition is presumed to be repeated minor traumata to the brain substance resulting in petechial haemorrhages; but here again there appears to be no documented evidence of such haemorrhages in Martland's article.[19] It would seem that the condition occurs rarely, if ever, in amateur boxers.

Wycis,[37] in reviewing the subject, states that after a certain period about 5% of boxers develop chronic progressive pathological changes and parkinsonian symptoms. Headaches, reflex disturbances, cranial nerve lesions and brain stem symptoms are described. A group termed 'berserks' appears to be of a severer grade, developing lack of restraint, irrationality and brutality. Antisocial or asocial behaviour is not infrequent and our Courts appear to deal with such cases every now and again. In this respect, it must again be stressed that the pre-traumatic personality, as in all head injuries, has a most important bearing on the post-traumatic symptoms.

According to Critchley,[6] when a fighter has experienced a groggy state repeatedly, fighting automatically without awareness of muscular movements, he is a candidate for punch-drunkness. Significantly, though, the symptoms and signs of this state may be associated with a perfectly normal electro-encephalogram. There would appear to be an overlap of the symptoms of post-traumatic parkinsonism and punch-drunkness. The latter, however, appears to be more often associated with emotional and psychic changes. The midbrain syndrome described by Russell and his co-workers emphasizes the site of traumatic change and it is the writer's contention that a lesser degree of these pathological disturbances is the underlying pathology of the boxer's post-traumatic encephalopathy, to a variable extent influenced by his pre-traumatic constitutional make-up.

SUMMARY

The writer, without entering the controversy of the pro's and con's of professional boxing, has discussed in some detail the problem of death in boxers, and has put forward his theory that, essentially, midbrain and brain stem injuries are the underlying causes of both mortality and morbidity in boxers.

A series of fatal cases is briefly discussed.

Prophylactic and preventive measures applied to the boxing industry are tabulated in the hope that their application may contribute to a lessening of the dangers to which boxers may be subjected.

OPSOMMING

Sonder om kant te kies in die geskil oor die voor- en nadele van beroepsbokswedstryde bespreek die skrywer die probleem van sterfgevalle onder boksers taamlik breedvoerig, en kom tot die gevolgtrekking dat beserings van die middelbrein en die breinstam die onderliggende oorsake van sterfgevalle sowel as morbiditeit by boksers is.

'n Reeks noodlottige gevalle word kortliks beskryf.

Profilaktiese en preventiewe maatreëls soos toegepas in die boksbedryf word uiteengesit in die hoop dat die toepassing daarvan kan bydra tot die vermindering van die gevare waaraan boksers blootgestel word.

The writer wishes to thank Dr. L. Meyerovitz for preparing Fig. 7.

He is also indebted to the late Mr. Bertie Sims, who kindly made available a copy of the South African Boxing Rules and Regulations.

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THE ROLE OF MEDICINE IN MODERN TIMES

WITH SPECIAL REFERENCE TO NUCLEAR MEDICINE*

S. F. OOSTHUIZEN, M.D.
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Mr. Chancellor, Mr. Vice-Chancellor, Members of the Council and Senate, Graduates, Ladies and Gentlemen: May I be permitted to start by thanking the University of Natal very sincerely for the great honour bestowed upon my esteemed fellow graduates and myself by the award of the honorary degrees. I think I can express the sentiments of all by assuring the University authorities that we are not only deeply conscious of the great honour, but that we are fully aware of the great responsibilities which membership of your illustrious institution has placed upon us.

I would like to convey congratulations to all those who have had degrees and diplomas conferred upon them to-day. It is a fitting occasion to congratulate specifically the newly qualified medical graduates who will have to live up to the reputation that they were the first medical graduates of this University. The eyes of the country and the world will be on them from now on. In welcoming them as members of the ancient profession I would like to emphasize the fact that membership of the profession of medicine brings great responsibilities, which only end with retirement or the grave.

The practice of medicine is no bed of roses, as the sick person is merciless in his demands upon those to whom his health is entrusted.

* Address delivered by Prof. S. F. Oosthuizen on the occasion of the graduation ceremony at the University of Natal on 29 March 1958, at Pietermaritzburg.

The added responsibilities of maintaining high standards of practice and an ethical code deeply steeped in the traditions of the past, will weigh heavily upon your shoulders throughout your professional life. To assist you in discharging your responsibilities, it may be useful to remember that there are potent reasons for being proud of the medical profession and to accept the additional responsibilities with a light heart. The profession of medicine has an honourable origin, it being the product of Greek intellect; it has revealed unparallelled solidarity in times of stress; it has advanced more rapidly than most other professions and it has delivered more free services to the sick poor than has any other profession.

At times when the criticism of patients, colleagues and others weigh heavily upon your souls, be not disheartened and remember that criticism and restlessness provide a firm foundation for progress. Greatness is often born out of suffering and strife. The ceaseless activity of comparatively insignificant ocean waves ultimately succeed in destroying the rocks on the shores. This illustrates the importance of continued activity as compared with inertia.

It is also well to remember that we have done nothing particular to render us immune to criticism and to provide us with the golden key to Paradise. As long as we believe in the importance of our mission as doctors, our efforts to survive will not have been in vain.

I also wish to congratulate the academic personnel for the outstanding results obtained at the first examination. This speaks well for the inspiring leadership of the Dean, Professor Gordon, supported throughout by his Principal, Dr. Malherbe and for the role played by other builders of the Faculty, e.g. Drs. George Gale and Alan Taylor.

Thank you also for your kind remarks about me, *Mr. Orator.*

I have chosen as the theme for my address *The Role of Medicine in Modern Times with Special Reference to Nuclear Medicine.*

With the advent of the atomic age, striking new methods were developed for application in the diagnosis, treatment and investigation of disease, quite apart from the boons to mankind which have resulted from the establishment of nuclear reactors, and the utilization of radioactive isotopes in agriculture and industry. It has, however, also brought new problems related to the potential dangers inherent in the use of radioactive sources. These dangers to Man of nuclear and allied rediations were known long before the first explosion of atomic bombs, but the developments which have resulted subsequently from the peaceful uses of atomic energy have greatly enhanced the importance of these dangers.

Radioisotopes are forms of matter which have been produced by altering the constitution of their nuclei, by bombarding a stable substance with particles from a particle-accelerating machine or nuclear reactor. The radioisotope so formed can be detected, and quantitatively measured by means of fairly simple instruments.

The medical uses of radioisotopes fall into 3 main groups:

i. Where the radiations emitted by the radioisotope, e.g. cobalt 60, are used to irradiate diseased organs in a way similar to the conventional methods of radiotherapy. Rays are directed through the aperture of a large shielded source on to the diseased site. The beam of rays so obtained is often superior to X-rays for the treatment of deep-seated tumours, as it is more penetrating and causes less discomfort to the patient.

ii. By selecting the appropriate radioisotope and administering it orally or intravenously, internal organs can be selectively irradiated. For example, phosphorus is taken up preferentially by bone so that diseased bone marrow can be selectively irradiated by administration of radioactive phosphorus. Iodine is almost exclusively taken up by the thyroid gland, and diseased thyroids can thus be treated with radioactive iodine.

iii. Perhaps the most important use of radioactive isotopes is in the diagnostic and research fields. Several diagnostic procedures using radioisotopes have virtually become standard procedures. The endocrinologist can hardly practise without using information obtained from tests with radio-iodine; the haematologist is concerned with the problems of iron metabolism as determined by radio-iron; physicians make use of studies of blood volume, circulation time, electrolyte balance and other functions by the use of radio-chromium, radio-sodium, radio-potassium and others. There is a great likelihood that mankind will also profit greatly from the knowledge gained about the growth of cancer through the use of radioactive materials as tracers.

A study of the results of pathological processes often precedes an appreciation of physiological error, and the isotope tool has proved to be invaluable in clarifying a great number of vexed problems.

The field of greatest usefulness to medicine of radioactive isotopes has thus been that of

applying tracer techniques to obtain a better insight in the function of the human body in health and disease.

The chances are that the use of radioactive isotopes will increase on all fronts, and that the problem of protection of workers as well as the public will assume greater proportions. There are advantages in the use of radioisotopes not only in medicine, but also from the economic point of view. It is, e.g. estimated that the savings achieved by U.S. industry, as the result of the use of radioisotopes, amount to well over 300 million dollars per annum, and it is expected that the 1,000 million mark will soon be reached. This appears to be a good return for the 15,000 million dollars spent on atomic energy since 1941 and will stimulate an ever increasing use of radioisotopes.

Health authorities are thus more and more faced with problems of adequate protection of the public as well as workers exposed to radiations. They are also concerned with the disposal of radioactive waste products. Hitherto the proportion of the population occupationally exposed has been small but circumstances are changing and the dangers are becoming more dispersed.

It is well known that the genetic effect of ionizing radiations is cumulative and, if not properly controlled, it may ultimately endanger the existence of the human race.

It is believed that, from the long-term hereditary or genetic point of view, a small exposure of radiation of the general population is just as serious as a high degree of radiation of a small group, the geatest danger being in the effect upon the reproductive organs. It is therefore the exposure of the general population which has been causing alarm in scientific circles.

There is, however, reason to believe that the potential hazard to Man of radiation has been grossly exaggerated. The topic became a popular one for non-scientific writers, and dramatic statements, without sufficient basis in fact, also originated from certain scientific quarters. There quickly followed a public awareness of the potential dangers to mankind by radioactive fall-out, and many scientific as well as non-scientific writers have put forward views about the present and future values of fall-out radiation based not only on knowledge of background radiation but also on unreliable deductions from calculations of radiation received from diagnostic and therapeutic procedures.

There has been widespread public concern about the seriousness of radioactive fall-out but, as a matter of fact, the daily amount of radiation received from this source is small in comparison with that received as the result of medical and other peaceful uses of atomic energy.

It is admitted that there is in existence a certain amount of background radiation from cosmic rays, from deposits of natural radioactive substances in the earth's crust and from radioactive isotopes in living tissues, but the amount of daily radiation is at present small. There is a difference of opinion amongst scientists whether or not fission and thermo-nuclear reactions should be entirely prohibited, notwithstanding the fact that the radiation from these sources remains slight. There is, however, some concern about the accumulation of strontium 90 in the bones of the human race as it predisposes to the development of bone tumours.

Public concern is not only due to the exaggerated statements about dangers of natural radiation and those resulting from the peaceful uses of atomic energy, but also about those resulting from ordinary X-ray procedures in hospitals and private practice.

There is evidence of adverse genetic effects, leukaemia, impaired growth of bone, premature ageing, etc. as the result of damage to the body brought about by radiations, but there is not sufficient reason for patients to refuse to undergo certain essential radiological examinations. The sharp decline in the number of pupil radiographers who fulfil an essential service to the community is also viewed with grave alarm by responsible hospital administrators, and there is great need for a public assurance that the dangers have been magnified out of proportion, and that the natural growth of an essential class of supplementary health personnel is being gravely endangered by irresponsible statements.

It is obviously the duty of the doctor to ensure that the health of the patient and coworker is safeguarded, and this is actually stringently observed by responsible authorities in South Africa and abroad.

There is, however, insufficient evidence to put into practice those dramatic suggestions which have contributed so much to existence of the present state of alarm. I am not minimizing the existence of the dangers, but plead for more realistic approach to the problem by the public and for the condemnation of alarmist tactics.

Competent authorities the world over are keeping a watching brief on developments, and the public should place their confidence in the scientists who devote their lives to a study of the problems which have become accentuated in the atomic age.

It is well to observe the greatest caution, but deep public concern, based on unreliable evidence, will prove a strong deterrent to future progress. Doctors and physicists are constantly on the look-out for safer methods of examination and treatment of patients and they will, through appropriate channels, keep the public properly informed of the true facts.

It is obviously the duty of radiologists and other physicians to make sure that X-ray examinations are reduced to a minimum and that technical advances which reduce the patient exposure be applied to practice whenever possible. Fortunately such technical developments are not slow in forthcoming.

Apart from the great advantages to mankind which have accrued from the peaceful uses of atomic energy and the disadvantage of potential harm, the developments in this new field have certainly created another special problem for the nations of the Western world in that the already existing shortage of scientists has been tremendously increased. This shortage is in existence in every field of endeavour related to atomic energy. Great efforts are being made in most countries, including South Africa, to make proper provision for the training of more personnel.

It is a matter of serious public importance that the services of scientists should receive greater and greater recognition and that it should be the policy of all progressive countries to ensure that adequate numbers are trained to serve mankind in the atomic age. It is always a good policy to invest in brains and to support the man with the idea.

I am pleased to say that the South African authorities directly concerned, e.g. the Atomic Energy Board and the C.S.I.R., appreciate the need to remain in step with the rest of the world, and that appropriate measures have been taken to establish a comprehensive atomic energy programme covering radiobiology and other fields, and enlisting the co-operation of the various universities in the matter. The need for radiation protection schemes, research, training of personnel, national and international co-operation and interchange of scientific information has been recognized and necessary steps have been taken for the implementation of a number of schemes.

It is possible that the potential hazard to Man of nuclear and other radiations may restrain intending trainees from coming forward. May I remind those hesitant for that reason, that our scientific forefathers never shirked their responsibilities, and many became martyrs for the benefit of science and humanity. We have reached a stage where reasonable freedom from danger for those who devote their lives to work in the nuclear energy field, can be assumed. The scarcity of accidents in modern atomic energy establishments bears eloquent evidence of the accuracy of this statement.

Apart from protection and training of scientific personnel, there is great need for further research in the biological and related fields. Fundamental research problems should preferably be 'farmed out' to competent workers at universities, whereas applied and reactor-research should, as far as possible, be centralized at national research institutions.

Particular attention should be paid to the provision of better facilities in the field of genetics, in accordance with the recommendations of WHO.

In fact, therefore, any national programme in the nuclear energy field should comprise a co-operative effort of all those who are concerned with the matter and this programme is, by the nature of the problem, so wide as to include universities, public health, national research and atomic energy authorities.

But notwithstanding the dramatic new developments and discoveries in the atomic era, and the emergence of a new branch of medical science, viz. nuclear medicine, which has revolutionized many concepts about disease and which has greatly supplemented the armamentaria of the doctor, there has been no fundamental change in the role which the medical man plays in modern times as compared with the past. The most important justification for the existence of the science of medicine, is the welfare and health of the patient. New discoveries may greatly facilitate the task of the doctor in preventing and curing diseases; they may even favourably influence the prognosis or outlook in certain serious, albeit age-old diseases; but no discovery, however startling it may be, can change the age-old doctor-patient-relationship, whether it be in general practice, specialist practice or the research laboratory.

Medical science is a warm personal science and it is concerned with human relationships. For this and other reasons, the role of medical science must be identified with the role of the doctor, who is the sole custodian of the health of his patient. All the startling developmen's

which have taken place in the immediate past, have not changed the primary function of the doctor and the ancient grip on his behaviour has in no way been relaxed. Medical science and the role of the doctor have become so deeply steeped in the traditions of the past that, in fact, powers almost equivalent to legal powers have been assumed.

The desire to help the sick has always been so great that the interchange of knowledge has continued when that of other sciences has stopped.

Because the medical man in his work as a doctor must be specifically identified with medical science, he emerges as the cornerstone in the never-ending battle against disease. Not only has he great responsibilities to his patients and, what is more important, to those who are not yet sick persons, but he has also great responsibilities towards his professional brethren and towards the Medical School where he received his training. Whereas in so many other professions, the professional man is answerable for his actions only to the ordinary courts of law, the doctor is also under the jurisdiction of his peers, to whom the legislature in many countries, including South Africa, delegated wide disciplinary powers by the establishment of statutory medical councils.

The sick person is an exacting taskmaster, the Medical Councils are strict in their efforts to uphold the honour and dignity of the profession, and the development of a professional brotherhood has resulted in unshakable traditions. The responsibility of the graduate to the Medical School is determined by a formidable father-child relationship. It is the duty of the medical man not to ignore what his father has done for him, and high ideals and fine words are insufficient and tantamount to lip service. It is the open-purse attitude which ensures most effectively that the fountain of knowledge does not dry up, and that the university can continue to discharge its responsibilities as the custodian of the ancient seat of learning.

It will, however, be wrong to assume that the responsibility is unilateral. The University as parent also has its responsibilities. It has become of great importance in modern times, that it should assume the role of leadership, ensure the availability of inspired teachers imbued with the spirit of service and provide an educational programme which includes adequate opportunities for reflection and study. In order to develop a taste for good food, one has to eat good food; in order to acquire the taste for good wine, one must drink good wine; and in order to acquire the best ingredients of scholarship and culture, one must associate with teachers of quality. If the graduate loses touch with these qualities available at his School, and fails to reap the benefits of post-graduate education, intellectual sterility will soon show its ugly face.

There must be the opportunity for graduates to continue the intellectual adventure which only a medical school can sponsor, where every problem should be an intellectual challenge and where the *status quo* is never accepted.

The role of the science of medicine, which is in fact synonymous with that of the medical man, is greatly influenced and modified by the responsibilities related thereto, whether these be in the field of medical education, medical research or simply in the maintenance of a lasting parent-child relationship.

Any relaxation of proper professional conduct produces a small furrow which soon becomes a mighty river in which so many of the essential values of professional life are drained away, leading to loss of confidence of the public in the professional man, to the detriment of all concerned.

Universities usually fulfil their function on the educational front, as it has long been appreciated that the wealth of an institution does not depend on the number of students, the bank balance or earthly possessions, but on the great names of teachers and students who are associated with it.

Medicine has a proud history of contribution to the arts, the sciences, statesmanship and service, and it has always backed scholarship and a high ethical code. It has often failed; but then it has been said that 'to die is no art, the weakest succeed, but to live is a great art because even the best fail to do it properly.' Psychological complaints are often ignored by busy practitioners and classified as hypochondria. The sufferer to whom such a complaint is just as upsetting as an organic one, invariably finds an ocean of comfort and sympathy in the arms of the quack, who does not hesitate to exploit the credulity of the public. Doctors should learn to be more diplomatic and start 'to remember the birthdays of their female patients without remembering their ages.'

In conclusion may I thank you once again, *Mr. Chancellor,* for having honoured me with the esteemed award, for inviting me to deliver the address at this auspicious and for many of us an historic and memorable occasion, and for having listened so patiently to what I have said.

ACUTE CAUSTIC SODA POISONING

OBSERVATIONS ON ITS TREATMENT

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and

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The present communication deals with 95 cases of acute caustic soda poisoning which have come under the care of the Thoracic Surgical Unit between August 1952 and December 1957.

There were 12 European cases and 83 cases of Coloured, Indian and African patients.

It is significant that 39 out of the 83 non-European cases were under 14 years of age, whilst only 2 out of the 12 European cases were under 14 years of age—an incidence of 47% and 17%. The youngest child was 11 months old and survived.

It is probable that caustic soda is both more frequently used and less carefully stored by non-Europeans: in a small house the only convenient cupboard is in the kitchen.

A number of acute cases were not referred to us, as they recovered rapidly with minor burns and no severe sequelae.

Grading of the strictures, as outlined in a previous communication, has been strictly adhered to,[1] viz. Grade 1, 2, 3, 4*a* and 4*b*.

Treatment: In the *acute stage*, treatment aims at dilution and neutralization of the corrosive, the control of shock, prevention of pulmonary complications, prevention of infection and maintenance of hydration and nutrition.

These subjects have not been improved on since our previous communication, which the reader is referred to for details.

PREVENTION OF STRICTURES

Dilatation still remains the mainstay in the treatment of strictures. In the present series it was attempted in all cases, and reluctantly abandoned when indicated.

INDICATIONS FOR ABANDONING DILATATION

1. *Poor Response to Dilatation.* In the present series, the main indication for abandoning dilatation has been a poor response to dilatation.

2. *Non-Passable Strictures.* Strictures have not been regarded as non-passable unless at least 2 attempts at dilatation have been unsuccessful. In these non-passable strictures, it is rarely possible to negotiate the obstruction by inserting several filiform ureteric bougies into the oesophagus. Pressure is then exerted on each bougie in rotation, until the lumen is established. Further dilatation may then be carried out with oesophageal bougies. A useful adjunct in these cases has been the passage of an infant's oesophagoscope or bronchoscope through the proximal stricture as far as the distal stricture. This has the great advantage of permitting the dilatation under direct vision until the size of the bougie exceeds that of the oesophagoscope or bronchoscope.

It has been our experience that the swallowing of a length of silk, the end of which is to be recovered from the gastrostomy wound, will not be successful if 2 attempts at negotiating the obstruction via the oesophagoscope have failed.

3. *The Age, Intelligence and Temperament of the Patient.* These considerations are particularly important if a satisfactory lumen has been established under anaesthesia, and is to be maintained by self-bouginage.

Each case has to be handled on its own merits. Rarely a child may swallow his own bougie religiously, whilst an adult may refuse to do so and be admitted repeatedly with food impacted at the site of the stricture.

4. *Perforation of Oesophagus* during dilatation of a tight stricture.

THE SURGICAL TREATMENT OF CORROSIVE STRICTURES

When any one or a combination of the indications for abandoning dilatation exists, some form of short-circuiting operation will be necessary. Since these operations are of considerable magnitude, it is most inadvisable to undertake these procedures without adequate and usually prolonged pre-operative care.

In our present series, gastrostomies were performed in 26 cases. Gastrostomy is a relatively minor procedure and has proved eminently successful. The general nutrition of these patients improved rapidly. With patience and well-balanced gastrostomy feeds, optimum conditions can always be obtained. It is unwise to try to shorten the period of rehabilitation after gastrostomy: 3 months is the shortest time devoted to feeding, and it has sometimes been lengthened to 6 months. Smooth and rapid

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recovery with good healing is the compensation.

The Gastrostomy. The routine 'inkwell' gastrostomy, done in the pre-pyloric portion of the stomach, has stood the test of time very well. It is important at this procedure to inspect and palpate the stomach in detail in search of severe burns. An hour-glass stricture may necessitate a gastro-gastrostomy. Narrowing of the pylorus, easily felt from the outside and confirmed by introducing the finger through the gastrostomy wound, requires pyloroplasty or a long-loop gastro-enterostomy combined with entero-anastomosis. The danger of stomach ulceration with the latter procedure is offset by the frequency with which afferent loop obstruction occurs with the subsequent gastropharyngostomy. Pyloric stenosis should, if possible, be diagnosed pre-operatively, and the gastro-enterostomy or pyloroplasty preceded by starvation for at least 48 hours or by gastric lavage, which is rarely possible.

Division of the arteries to the stomach at this stage is inadvisable. If the gastropharyngostomy is done within a few weeks, fresh vascular adhesions will be a nuisance; whereas if it is done after an interval of several months, dense adhesions with no anatomical pattern will delay the mobilization of the stomach.

The extra safeguard of bringing the gastrostomy tube through a hole in the greater omentum is not essential. In fact, it makes the subsequent mobilization of the stomach more difficult.

Oesophago-Gastrostomy and Gastro-Pharyngostomy. A delay of several months is often necessary after the gastrostomy has been performed. During the waiting period, diversional occupational therapy and physiotherapy are instituted. It is remarkable that, even in suicidal cases, the morale of the patient is high because he knows that after the operation he will be able to swallow 'the normal way' again. Intensive and careful feeding with a well-balanced diet is carried out during this preparatory interval.

A week before the operation, barium studies of the oesophagus and stomach are made. It is important to have a good view of the pharynx and cervical oesophagus in these films. The level of the highest stricture is marked on the patient and any deformity of the stomach or delay in emptying is noted. The gastrostomy wound must be healthy. This is the part of the thoraco-abdominal incision that often fails to heal by primary intention.

THE OPERATION

This is preceded by oesophagoscopy. The level of the stricture is again noted. The mucosa is inspected and the oesophagus and pharynx are packed with gauze strip. In strictures above the level of the manubrium sterni, the exact level of the obstruction can be accurately plotted by palpating the tip of the oesophagoscope.

Positioning and Towelling of the Patient. Whilst the patient is in the supine position, suction is applied to the gastrostomy tube during its withdrawal. A deep mattress suture is inserted through the gastrostomy wound to prevent the inevitable soiling of the towels which will occur if it is left patent.

The intravenous drip is inserted in the right forearm, and the patient is turned on his right side. A sandbag is placed under the neck. The left side of the neck must be shaved.

The left arm is towelled and bandaged firmly, so that it can be freely mobile in a sterile field. The aim is to obtain clear fields for both the thoraco-abdominal and the cervical approaches.

The Approach. An 8th intercostal thoraco-abdominal approach, carried forward to encircle the gastrostomy, gives excellent exposure to the stomach and lower oesophagus. In adults, this approach may have to be supplemented by a 4th intercostal approach through the same skin incision in order to do a supra-aortic intrathoracic anastomosis. The same additional 4th intercostal approach may be needed to dissect out the pathway through the dome of the pleura, when the stomach is carried through for a cervical anastomosis. It is unwise to try this dissection from the level of the 8th intercostal space. Shingling of one or two ribs above the 4th intercostal approach may be necessary to give adequate exposure.

The diaphragm is divided from the 8th costal cartilage to the hiatus, and the stomach is freed from all its vascular attachments to the posterior abdomen except the gastro-epiploic. The gastrostomy opening is separated from the abdominal wall and securely closed in 2 layers.

The oesophagus is freed from the mediastinal connective tissue to which it may have become firmly fixed, up to the level of the aortic arch. The opposite pleura may be opened at this stage. This is no great complication if it is noted and the anaesthetist is warned; but it may be that the right lung is also adherent to the scarred oesophagus, and may be injured if not recognized.

One or Two-Stage Operation. The fundus of the stomach is now brought up, and its laxity and colour assessed. If the anastomosis is to be done behind or just above the aortic arch, there is no reason to put it off to a second stage: there will be no tension to endanger union.

The oesophagus itself need never be divided or removed, and this avoids what is sometimes a difficult manoeuvre from fixity of the oesophagus to the aortic arch. It has never caused trouble except once, when persistent granulations kept on bleeding even after 5 years. On one occasion it produced discomfort due to food stoppage in the blind end.

Anastomosis of the fundus to the oesophagus below the aortic arch will risk, according to Allison and to our experience in one case of achalasia of the cardia, peptic erosion at the suture line. Supra-aortic suturing has never, in this series, shown symptoms or endoscopic evidence of peptic irritation.

If the fundus is to be carried up into the neck, the mediastinal pleura is divided parallel to and behind the subclavian artery and the aortic arch, through a 4th intercostal approach. The scarred oesophagus is exposed and a tunnel very easily made with the finger running up along the oesophagus. Unless the mediastinal incision is deepened down to the oesophagus, lifting up both the subclavian and the left carotid arteries, this tunnelling may be very difficult. It is at the origin of the subclavian artery that the thoracic duct may be injured. The tunnel is easily stretched until it takes 2 fingers comfortably: at no time has the first rib prevented this, although Sibson's fascia has occasionally needed division behind the subclavian artery. The neck is entered medial to the carotid sheath and to the scalenus anterior, and the finger burrows its way bloodlessly to the prevertebral space next to the thyroid gland.

A mediastinal incision between the 2 vessels does not allow as easy or as roomy an approach.

The arm and the shoulder are now brought down and an incision made in the skin along the left sternomastoid. The oesophagus is exposed by dividing the middle thyroid vein: the gauze pack easily identifies the pharynx and lifts up the larynx. A curved sponge forceps is passed down the tunnel into the chest and the fundus of the stomach grasped and pulled up.

At this point the colour and tension of the stomach must be carefully judged, to decide whether anastomosis to the oesophagus or pharynx is done at once. If there is any doubt, it is important to give the stomach the benefit, and to attach it to the skin of the neck at the level of the cricoid or higher, and also anchoring it to the longus colli, sternomastoid and scalenus anterior. Acute dilatation of the stomach is common, and will cause tearing at the sutures. It is essential, therefore, to open the stomach in the neck and pass a wide tube into it down to its lower end. The second stage, turning the open mouth in and joining it to the gullet, is easily done 2 or 3 weeks later. A premature one-stage operation risks a tedious and even dangerous fistula.

The stomach is stitched to the neck muscles in such a manner as to close off any part of the tunnel to the entry of air to the chest.

In this series, 14 pharyngogastrostomies and 7 oesophagogastrostomies have been performed. In a further 3 cases, jejunum had to be used for the anastomoses.

In the 21 cases where the stomach was used for anastomosis, fistula formation occurred in 3 cases, one of which terminated fatally. In the 3 cases mentioned previously, where jejunum was used, intrathoracic fistula formation and death resulted in the first case. In the second patient, the jejunum, which had been placed subcutaneously anterior to the sternum, sloughed completely. Dilatation of the original oesophagus was resumed with very moderate success. In the third case, a fistula formed in the neck. The fistula was successfully closed.

Of the 24 short-circuiting operations (14 + 7 + 3), 5 deaths resulted. Two of these deaths were due to fistula formation; one death was due to haemorrhage from the subclavian vein. In the 4th fatal case, death was due to heatstroke. The fifth case died suddenly 10 days after the second-stage gastropharyngostomy, probably from pulmonary embolism.

In the present series, intrathoracic fistula formation carried a 100% mortality. No deaths resulted from fistula formation in the neck. Complete sloughing of a subcutaneous jejunum did not produce death.

SUMMARY

1. This communication deals with some of the more important observations that have been made on the treatment of acute caustic soda poisoning between August 1952 and December 1957.

2. Certain aspects of the subject have been more fully described in previous papers, viz.:

(*a*) The technique of high oesophago-gastric anastomosis.[2]

(*b*) Acute caustic soda injuries of the oesophagus.[3]

(*c*) Caustic strictures of the oesophagus.[4]

(*d*) The treatment of caustic strictures of the oesophagus.[1]

3. Dilatation, repeated at regular intervals, and sometimes persisted with for years, still remains the mainstay in the treatment of strictures.

4. The indications for abandoning dilatation are outlined.

5. The value of prolonged gastrostomy feeds before the major 'short-circuiting' is stressed.

6. Some important aspects on the technique of gastropharyngostomy and gastro-oesophagostomy are discussed. Intra-thoracic fistula formation has carried a 100% mortality.

7. Five deaths have resulted after 24 short-circuiting operations—a 21% mortality. In a previous communication[1] the mortality rate was 20%.

Probably some of the deaths in the present series could have been avoided.

No gastric perforations remote from the site of the anastomosis occurred in the present series.

A total of 247 cases have been under our care since July 1948; 64 came to operation of oesophageal by-pass, with a mortality of 13 (20%).

We are grateful to Dr. I. Frack, Medical Superintendent of Baragwanath Hospital (and to the late Dr. J. D. Allen, former Medical Superintendent), for allowing us to investigate and treat many of their cases. Our thanks are also due to the anaesthetists, the Theatre Sisters and the Nursing Staff of Baragwanath Hospital, without whom this work could not have been done.

OPSOMMING

1. Hierdie bydrae handel oor sommige van die vernaamste waarnemings wat gedoen is tydens die behandeling van akute bytsoda-vergiftiging gedurende die tydperk Augustus 1952 en Desember 1957.

2. Sekere aspekte van die onderwerp is volledliger beskryf in vorige bydraes:

(*a*) Die tegniek van hoë slukderm-gastro-anastomose.

(*b*) Akute bytsodabeserings van die slukderm.

(*c*) Bytsoda-vernouing van die slukderm.

(*d*) Die behandeling van bytsoda-vernouing van die slukderm.

3. Die indikasies wat dit nodig maak om van verwyding af te sien, word uiteengesit.

4. Die waarde van langdurige gastrostomiese voedings vóór die groot ‚kortsluiting' word beklemtoon.

5. Sekere belangrike aspekte van die tegniek van gastrofaringostomie en gastro-oesofagostomie word bespreek. Die vorming van fistels binne in die borskas het 'n sterftesyfer van 100% tot gevolg gehad.

6. Vyf sterfgevalle het gevolg na 21 kortsluitingsoperasies—'n sterftesyfer van 24%. In 'n vorige mededeling was die sterftesyfer 20%.

Dit is moontlik dat sommige van die sterfgevalle in die huidige reeks vermy kon gewees het.

Geen maagperforasies, verwyder van die plek van die anastomose, het tydens die huidige reeks voorgekom nie.

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ACUTE POLIOMYELITIS*

A STUDY OF THE CLINICAL MANIFESTATIONS OF FIFTY CASES

SEEN AT THE CHILDREN'S HOSPITAL, JOHANNESBURG, DURING THE 1948 EPIDEMIC

WITH SPECIAL REFERENCE TO THE MANAGEMENT IN THE ACUTE PHASE

J. L. BRAUDO, M.B., B.CH., M.R.C.P. (EDIN.)
Johannesburg

(*Continued from p.* 454)

V. PROGNOSIS: IMMEDIATE AND ULTIMATE

The problem of prognosis in acute poliomyelitis involves several considerations:

i. What is the mortality?
ii. Will paralysis appear?
iii. Will an initial paralysis advance?
iv. Will the paralysis be permanent?

i. MORTALITY RATE

In regard to the first question the following figures have been ascertained. In this small series comprising cases under 14 years of age the number of deaths was 4 of 50 cases (8%). In the 1947–8 Johannesburg epidemic, 443 cases of the 623 notified occurred in children under the age of 14 years. In this group there

* The References will be published at the end of the concluding article in this series.

were 22 deaths with a mortality of 5%. The figures in the 1944–5 Johannesburg epidemic were somewhat different. Of the 174 cases reported, 144 occurred in the 0–14 year age group with a mortality rate of 12% in the latter group as compared with 13% for the total group. Hence the mortality rate was considerably lower in the 1948 epidemic. The mortality rates in various parts of the world during the same period, as well as those in earlier epidemics, are compared with those of the Johannesburg epidemics in Table 11

TABLE 11: MORTALITY RATE IN VARIOUS EPIDEMIC

Date	*Author*	*Number of Cases*	*Total Percentage Mortality*	*Percentage Mortality (0—11 years)*
1905	Wickman (Sweden)[4] ..	868	17%	12%
1910	Ruhräh and Mayer[32] (Massachusetts) ..	1,216	8%	—
1943	Agius *et al.*[31] (Malta) ..	426	3%	3%
1944	Millar[5] (Johannesburg) ..	174	13%	12%
1948	Millar[5] (Johannesburg) ..	623	4%	5%
1950	Braudo (Johannesburg) ..	50	—	8%

In Wickman's series of 868 cases the total mortality rate was 17%—in patients below 11 years 12% and between the ages of 12 and 20 years 29%. In the Massachusetts epidemic of 1910 the mortality rate was 8% in a series of 1,216 cases. In both these epidemics the mortality rate was considerably higher in the older age group. Of the 426 cases reported in the 1942–3 Malta epidemic, 15 cases died (a mortality rate of 3%). However, all the cases were under 10 years of age, with the vast majority under 5 years. This feature probably accounted for the very low mortality rate. All these authors agreed that the mortality rate was higher in the older age groups, i.e. in patients over 12 years of age. Furthermore, it seemed as if the overall epidemic mortality rate was somewhat lower now than it was before World War I. However, death rates varied greatly in epidemics and in the Indian epidemic described by McAlpine,[61] a mortality rate of 37% was recorded. This outbreak was confined to physically active adult soldiers, which probably accounted for the very high death rate. The deaths in poliomyelitis usually occurred in the bulbar or bulbo-spinal group of cases. They were due to involvement of the vital respiratory and vasomotor centres in the medulla alone or in combination with paralysis of the peripheral respiratory and swallowing musculature. Consequently, the prognosis in these varieties of the disease was much more serious than in the spinal paralytic variety. Death, when it came, usually occurred between the fourth and eighth days of the illness, but in some cases within 12–24 hours of the onset of the disease. In this series one case died on the first day of the illness, one on the fifth and 2 on the eighth day.

Age. The death rate was usually higher in the older age group. In the 1947–8 Johannesburg epidemic, however, most of the deaths occurred in the 2–10 year age group, which showed the greatest incidence of the disease. There were 16 deaths among 306 cases, a mortality rate of 5% which was 1% higher than the mortality rate of the whole epidemic.[5]

Sex. Of the 4 deaths in this series, 3 were males and one female. Of the 14 cases with bulbar involvement, 7 were males and 7 females. Of the 9 seriously ill cases, however, 6 were males and 3 females. Grulee and Panos[29] noted that 17 of 20 tracheotomized cases were boys and 70% of 113 cases belonging to the bulbar group were male. Furthermore, 85% of the children requiring respirators were boys. It therefore seemed reasonable to be more guarded in the prognosis of cases in the male bulbar group.

Prodromata. An attempt will now be made to correlate the clinical pattern of the disease with the final outcome. The diphasic disease pattern was noted in 5 cases of the bulbar and bulbo-spinal group; 3 died and one made a fair recovery. In the straggling pattern there were 4 cases with no deaths and only one poor result. In the invasive type, however, there were 5 cases with one death and 4 good results.

These figures showed that the greatest mortality rate occurred in the diphasic pattern. Nissen[62] noted that the dromedary or diphasic type of poliomyelitis had a bad prognosis. He observed that 76% of his paralysed cases had such pre-paralytic manifestations whereas only 29% of the non-paralytic cases were of the diphasic type. In this series 11 of 33 cases (33%) were of the diphasic variety in the bulbar, bulbo-spinal and spinal paralytic groups. However, only 3 of 17 cases (18%) of the diphasic variety appeared in the non-paralytic group. It would appear, therefore,

that cases of the dromedary or diphasic pattern of poliomyelitis tended to be more serious than those of the straggling or invasive patterns.

Clinical Features. The clinical features of 3 of the fatal cases were very similar. Unfortunately, the fourth case was admitted in a moribund state and the early clinical findings are not available. All 3 showed marked encephalitic symptoms which increased with the extension of the disease. They were characterized by extreme apprehension, restlessness, twitching of muscles, tremor of the outstretched hands and hyperactivity. Marked neck and spinal stiffness together with cold, moist extremities were also present. They all showed secondary rises in temperature and persistent, marked tachycardia. The blood pressure was increased, a small pulse pressure being noted in 2 cases. Two patients also developed ectopic heart beats before their demise. These cases remained conscious, with extreme anxiety and alertness, right up to the last breath. Profound coma did not occur in any of the fatal cases. Marked nystagmus of both the horizontal and vertical variety was recorded in 2 of the fatal cases.

Paralysis of the diaphragm and intercostal muscles was not necessarily of grave importance, although present in 3 of the 4 fatal cases in this series. Peabody *et al.*[30] and Draper[18] stressed that increasing alertness and apprehension on the part of the child with an existing paralysis was of grave significance. None of their profoundly stuporous cases died. They also noted that children with paralysis of the diaphragm and intercostal muscles recovered despite severe involvement. All their fatal cases had paralysis of one or both deltoid muscles together with the respiratory involvement. The clinical picture of the severely ill child has therefore not changed appreciably in the past 40 years.

Death might occur in cases with bronchial aspiration superimposed upon pharyngeal paralysis, or asphyxia due to laryngeal palsy. These cases showed extreme cyanosis, marked recession of the lower costal margins, tachycardia and tachypnoea and eventually death. Prompt measures, e.g. tracheotomy, aspiration, oxygen, etc. could save the patient's life. The signs and symptoms of hypoxia were very important since death ensued if the anoxia was severe enough. There were 4 stages.

First Stage: Restlessness, apprehension, anxiety and sleeplessness.

Second Stage: Florid appearance, increasing pulse rate and blood pressure, respiratory effort, aphasia and a tendency to speak fewer words in one breath.

Third Stage: Dyspnoea, cyanosis, rise in temperature, some confusion and occasional panic reaction.

Fourth Stage: Cyanosis, circumoral twitching, failure to answer questions, delirium, coma and terminal shock.

Bacterial pneumonia secondary to atelectasis and poor respiratory excursion was a grave complication before the days of modern antibiotic therapy, but is no longer a threat to life.

Were the CSF findings and blood counts of any prognostic significance? The CSF pressure was raised in 3 cases, markedly so in 2 (200 mm. and 210 mm. CSF respectively) and normal in one. The cell count was very high in 2 (478 and 258 cells per c.mm. respectively) and slightly raised in 2 (35 and 28 cells per c.mm. respectively). The protein content of the CSF was 100 mg. per 100 c.c. in one case and normal in the other 3, but the sugar content was raised in 3 cases (120 mg., 90 mg. and 85 mg. per 100 c.c.). Blood counts in 2 of the fatal cases revealed a polymorphonuclear leucocytosis with normal sedimentation rates.

Raised pressure and sugar values in the CSF were most common in the bulbar and bulbospinal cases, especially the very severe ones and rarely occurred in the non-paralytic and spinal paralytic groups.

Draper[19] stated that contrary to the opinion usually held, the spinal fluid undoubtedly offered some prognostic indications. Counts taken in the first 12–18 hours of the disease might be of some value, but not those taken after 24–36 hours. Cases with cell counts of 200–500 per c.mm. in the very early phase frequently developed paralysis of a severe nature, whereas counts of less than 100 per c.mm. in the first 12–18 hours rarely developed muscular weakness. With few exceptions his fatal cases showed counts of over 700 or 1,000 cells per c.mm. Most authors do not agree with Draper's comments on the CSF and even in this small series the fatal cases did not have counts above 500 cells per c.mm. The raised sugar content and high CSF pressure may be of serious prognostic importance. No reference to the prognostic significance of these 2 features could be found in the literature.

ii. THE APPEARANCE OF PARALYSIS

The next question to be discussed is whether paralysis will appear. It is impossible to say that palsy will or will not occur when the

disease is diagnosed at its inception. In this series 33 of 50 cases (66%) showed paralysis at some stage of the disease. Five hundred cases occurring in the 1947–8 Johannesburg epidemic were analysed. Two hundred and fifty-five cases (51%) had paralysis of one form or another. One hundred and sixty-three cases were described in the 1947–8 South Australian epidemic; of these 103 (64%) were of the paralytic variety.[17] All the cases described in the 1905–17 period in Johannesburg were of the paralytic type. In the 1943 Malta epidemic all the cases had some form of paralysis.[31]

The chances of paralysis occurring in a given series of poliomyelitis cases clinically diagnosed were therefore greater than 50%. This fact in itself was of great prognostic significance. Paralysis most frequently appeared in the first 4 days of the illness, but might come on as late as the twelfth day. In this series 29 cases occurred in the first 4 days, one on the fifth day, one on the seventh and 2 on the eighth day. In both the older epidemics,[30, 33] the time intervals between the onset of the disease and paralysis were very similar and the likelihood of paralysis diminished rapidly after the fourth day and was minimal after the eighth day of the disease.

Can the prodromata, symptoms and signs and laboratory findings help in prognostication? The prodromal symptoms were the same in all types of poliomyelitis but the pattern differed.

The only gross difference between the 2 varieties was noted in the diphasic and straggling forms. The diphasic pattern was commoner in the paralytic and the straggling pattern in the non-paralytic cases. The percentage figures for the invasive group were the same. The number of cases is too small to be of statistical value, but the trend indicates that paralysis appears more often in the diphasic pattern, which occurred almost twice as often in the paralytic variety. Forty-one per cent of the non-paralytic group had the straggling pattern whereas only 27% showed this form in the paralytic group. The invasive form of the disease was seen in 41% of the non-paralytic and 39% of the paralytic cases, occurring with about equal frequency in both varieties.

Symptoms and signs were not strictly comparable in both groups since the cases were not seen on the same day of the illness. Furthermore, those complaining of paralysis on admission had to be assessed in retrospect. The frequency of the important symptoms and signs will be found in Tables 9 and 10.

Symptoms. The only significant difference as regards symptomatology was the presence of limb pain in 8 cases of the paralytic group and only 2 cases of the non-paralytic group, a ratio of 4:1. All the other symptoms apart from weakness of some specific musculature, which occurred in 17 cases, occurred with about the same frequency in both groups.

Peabody *et al.*[30] stated that they had found no absolute way of anticipating paralysis. Occasionally a patient complained of pain in a limb which was subsequently lamed. In this series the subjective complaint of pain in specific limbs only corresponded to the subsequently paralysed ones in 3 cases, whereas 4 other cases complained of pain in all limbs with weakness confined to only one leg in 3 and one leg and both elbows in the fourth one. One case with pain in the left leg and the abdomen later had paralysis of the right deltoid and the right diaphragm. None of the bulbar cases had pain in the distribution of the palsied muscles, viz. face, throat or eye. Faber[63] says the sore throat is of neurogenic origin because no swelling or exudate is present and in 5 cases representing 25% of cases complaining of sore throat, paralysis ensued. Only 3 of 50 complained of sore throat in the invasive phase and one belonged to the bulbar group. Both groups had a large number of cases with pain in the neck, back or both. Pain in the neck only was associated with 2 cases of bulbar palsy, whereas pain or stiffness of the back was associated with paralysis of both upper and lower limb musculature. Pain or stiffness of the back was a common complaint in the non-paralytic cases.

Although there was some correlation between the site of the pain and subsequent weakness in several cases, it was by no means constant. The presence of pain in a specific area, part or all of which later became paralysed, was noted in 7 cases in the spinal paralytic group. In 3 other cases its presence was of no significance.

Signs. The only signs showing marked differences were tremor, hypertension, Hoyne's sign and hypo-reflexia. Although they were found in some of the non-paralytic cases, the incidence was 3 times greater in the paralytic group in respect of tremor, 3.5 times in respect

of hypertension, 4 times in respect of Hoyne's sign and 15 times in respect of hypo-reflexia.

Pollock[64] stated that deep reflexes were absent in over 90% of cases destined to be paralysed, whereas they were absent in only 3% of cases examined and not destined to be paralysed. Furthermore, the superficial abdominal reflexes were present in only 3% of cases wherever the lower extremities were paralysed or destined to be so. In this series abdominal reflexes were only absent when the abdominal musculature itself was paralysed. Peabody *et al.*[30] stressed the fact that disappearance of tendon jerks was not an infallible sign of paralysis. Hypo-reflexia or areflexia was seen in only one case of the non-paralytic group but was found in 15 cases of the spinal group, usually after paralysis had set in. Several instances of areflexia of one tendon jerk with hyper-reflexia in several other deep tendon reflexes were noted. The presence of these signs may therefore point to the possibility that paralysis may develop. Neck and spinal stiffness, hyper-reflexia and reddening of the pharynx were frequently present in both paralytic and non-paralytic groups. A sudden rise in temperature and pulse rate was liable to be accompanied by weakness in a case that had hitherto been non-paralytic, or extension of the palsy already present.

The Effect of Trauma and Physical Activity. The relationship of operations, inoculations, injections, fatigue and trauma to paralysis in poliomyelitis will now be considered. Operations such as tonsillectomy and dental extraction[65] especially the former, were contraindicated during an epidemic of poliomyelitis because they predisposed to the bulbar variety of the disease. This was the opinion of authorities all over the world. It would be interesting to analyse some of the facts. In this series 2 cases developed poliomyelitis within 3 weeks of tonsillectomy. One developed the non-paralytic form of the disease and the other the bulbo-spinal variety. Both cases made complete recoveries. Aycock[66] reviewed the literature at length and showed that in some 170 cases in which the operation had been followed within 30 days by poliomyelitis, the bulbar or bulbo-spinal variety occurred in 121 cases (71%). Anderson[67] reviewing the 1943 Utah epidemic, concluded that the incidence of poliomyelitis in subjects of recent tonsillectomy was nearly 3 times greater than that in the general child population and that the incidence of the bulbar form was 16 times greater. On the basis of findings in the 1937 Toronto epidemic, it was estimated that of children in the general population between the ages of 3 and 12 years, 3 per 1,000 developed poliomyelitis. Among these children of the same age group known to have had tonsillectomy at the height of the epidemic, 5 per 1,000 developed poliomyelitis. In the extra 2 per 1,000 the disease was of the bulbar type.[68]

There was experimental evidence to show that if the virus were present in the pharynx at the time of, or shortly after, operations on the pharynx, clinical poliomyelitis would be precipitated and was likely to be of the bulbar type. Sabin[69] injected a suspension of virus around the tonsil in a series of 20 monkeys. Of this series 18 developed poliomyelitis, 13 cases of the bulbar type. It was assumed that the operation opened up a portal of entry for the virus already present in the pharynx. The afferent neurones which the operation would affect would be the sensory nerves in the neighbourhood of the trauma, i.e. cranial nerves V, IX and X; but Faber and Silverberg[70] in a number of poliomyelitis examinations found that these neurones were affected in almost all cases, whether the disease was bulbar or spinal. It was possible that the surgical trauma exposed the efferent motor nerves of the pharynx and the virus might ascend these axons directly to the nuclei in the medulla. If this were so then one would expect surgical trauma to other muscles, coincidental with incipient poliomyelitis, to lead to paralysis of these muscles. This problem will be discussed later when inoculation and injections in relation to paralysis in poliomyelitis are considered.

Most of the patients developing poliomyelitis after tonsillectomy recover completely, but many fatalities have occurred, e.g. the famous family in which 3 deaths occurred in one family following 5 simultaneous tonsillectomies performed on the same day.[71] We can therefore conclude that tonsillectomy is a dangerous procedure during epidemic periods of poliomyelitis and should be postponed. Cunning[72] summarized the present position as follows:

'After 10 years' study of this subject, reviewing over 17,000 poliomyelitis cases and 35,000 tonsillectomy cases, I am still of the same opinion as I was one year ago—that no definite causal relationship between tonsillectomy and poliomyelitis has been

established and I do not believe that tonsillectomy be postponed indefinitely simply because the summer months are the months during which poliomyelitis is prevalent'.

Nevertheless he stated:

'If there is a distinct rise in the poliomyelitis rate, bordering on epidemic proportions, all elective operations should be postponed'.

In this series there were no instances of prophylactic anti-pertussis or anti-diphtheria inoculations 3 months before the onset of the disease. Three cases, however, had injections within the recognized period of 0–30 days of the onset of the disease. Case 32 of the spinal paralytic variety received 3 penicillin injections into the right buttock and developed paralysis of the right leg 6 days later. Case 33 of the spinal-paralytic group (a brother of Case 32) had 3 penicillin injections into the left buttock and developed paralysis of the left lower limb 3 days later. Case 48 of the spinal-paralytic group was admitted on 9 April 1948 with a painful right upper arm which was diagnosed clinically as osteitis of the upper end of the right humerus. He was given numerous penicillin injections into both buttocks and thighs over a period of 2 weeks and the osteitis of the arm resolved. Two weeks later he developed poliomyelitis and a weakness of the muscles of his left shoulder girdle. In this case the paralysis did not coincide with the disease in the right arm and did not affect the other sites of numerous intramuscular injections, viz. the right shoulder and both buttocks.

McCloskey[73] noted 53 cases in which an injection of diphtheria toxoid or pertussis vaccine or both was accompanied by severe paralysis in the injected limb. There was also evidence that recent inoculation of pertussis vaccine alone or in combination with diphtheria toxoid was more likely to be followed by paralysis than recent inoculations of diphtheria toxoid alone. Furthermore, the severity of those cases following pertussis vaccine was greater. Forty-one of the cases received their last inoculation within one month of the onset and 12 between 1–3 months. Most of the paralyses occurred within 7–11 days of the injection, i.e. within the accepted period of the incubation period of poliomyelitis, and there was an equally striking localization of severe paralysis to the limb inoculated nearest to 7–11 days before the onset of the illness.

McCloskey's findings have been confirmed by other workers.[74, 75] Hill and Knowelden[76] in a statistical investigation in England and Wales in 1949 came to the following conclusion:

'The distribution of the bodily sites of paralysis was quite normal in children who had been inoculated within the month preceding the onset of their illness. In this group paralysis in the arms was just as frequent as paralysis in the legs and the left arm showed paralysis more often than the right; in children without recent injections the 2 arms were equally affected and the legs were affected 2–3 times as often as the arms. The left arm is the most common site for inoculation in England and Wales. There was no evidence whatever that inoculations carried out in the past have any effect at all upon the incidence or localization of the paralysis. Paralysis in the limb of recent injection followed both inoculations with A.P.T. and combined A.P.T. and pertussis antigens'.

Very few clinical cases of paralysis localized to traumatized limbs, other than inoculated ones, have been reported and the occasional association between local trauma and eventual paralysis is not convincing clinically[77] or experimentally.[78] In this series, Case 43 of the spinal paralytic group fell and hurt her left leg. Despite some improvement during the following 2 days, definite weakness was noted on the fourth day. In this particular case it was difficult to assess the role of pain due to the disease itself, restricting movement, and that due to trauma. Furthermore, it is possible that the limb was weak to begin with and resulted in the fall.

Why do related cases of poliomyelitis not occur in extensive controlled trials of pertussis immunization? Why is the association between inoculation and paralytic poliomyelitis very rare in America where a huge proportion of infants is immunized and poliomyelitis is very common? Is intramuscular injection more potent than the subcutaneous route? Is the impression that pertussis vaccines are much more dangerous than diphtheria toxoid preparations actually justified? All these problems and the relation between inoculation and subsequent paralysis have still to be settled. A study of the literature at this stage supports the contention that prophylactic immunization should be withheld for any period in which there is a high incidence of poliomyelitis in the community concerned. Diphtheria immunization is so important, that only very clear evidence of harmful sequelae should be allowed to interfere with the policy of universal immunization. On the other hand, the usefulness of pertussis vaccine has not yet been fully established. It should not be advocated during epidemic periods.

(To be continued)

NOTES AND NEWS : BERIGTE

Dr. B. A. Bradlow, who has spent 3 months visiting clinics and hospitals in the United Kingdom and on the Continent, has returned to Johannesburg and has resumed his practice.

* * *

Mr. D. J. Retief, F.R.C.S. (Eng)., orthopaedic surgeon, has moved to 203 Osler Chambers, 215 Jeppe Street (Corner Delvers Street), Johannesburg. (Telephones: *Rooms*: 23–1718; *Residence*: 44–3242; *Emergency*: 22–4191.)

* * *

Mr. Felix Machanik, orthopaedic surgeon, has changed the address of his rooms to Room 5—West Wing (3rd Floor), Clarendon Centre, Clarendon Circle, Johannesburg. The new telephone number is 44-0956.

Mnr. Felix Machanik, ortopediese chirurg, het die adres van sy spreekkamers verander na Kamer 5—Westelike Vleuel (Derde Verdieping), Clarendon Centre, Clarendon-Sirkel, Johannesburg. Die nuwe telefoon nommer is 44-0956.

* * *

Prof. J. H. Louw, Head of the Department of Surgery, University of Cape Town, has received a Carnegie Travel Award, which will enable him to visit surgical teaching centres in America.

Professor Louw, accompanied by Mrs. Louw, will leave early next year.

He will have an oportunity to pay attention to his special field of interest, viz. paediatric surgery, during his stay in Boston. He will also visit clinics specializing in vascular surgery.

* * *

Prof. G. A. Elliott, Head of the Department of Medicine, University of the Witwatersrand, Johannesburg, has been awarded a W.H.O. Fellowship to visit Great Britain and the United States of America to study *The Effect of Radiation on Man*, with emphasis upon the clinical and genetic effects on the individual and on the community. The clinical study will include particularly the effects on the blood and the bone marrow and the present status of the position of marrow replacement.

Prof. G. A. Elliott

Professor Elliott plans to leave in September this year. His tour will include most of the well-known institutions in Great Britain and the U.S.A. as far west as Chicago. On his return to South Africa early next year, Professor Elliott will travel overland (by Land Rover) via France, Switzerland, Italy, across the Mediterranean to Tunis, through Libya, Egypt, the Sudan, Uganda and the Federation as his last stop on the way back to Johannesburg. In the course of this tour across Africa, Professor Elliott will visit Medical Schools and Institutions en route.

Mr. Wilfred Kark, F.R.C.S., has changed his consulting rooms from 33 Jenner Chambers to 46 Lister Buildings, Jeppe Street, Johannesburg. The telephone numbers remain unchanged, viz.: *Rooms*, 23-9560; *Residence*, 41-1605.

* * *

The South African Council for Scientific and Industrial Research, which is sponsoring a Pneumoconiosis Conference in Johannesburg, has announced that the Conference will be held from 9–24 February 1959.

The President is Prof. S. F. Oosthuizen. The Secretary-General is Dr. A. J. Orenstein.

A provisional programme has now been issued. Copies may be obtained from Dr. A. J. Orenstein (*Secretary-General*), P.O. Box 4788, Johannesburg.

* * *

CHANGE OF DISTAQUAINE V 120 MG. AND 240 MG. TABLET STRENGTHS

Phenoxymethylpenicillin (Distaquaine V, penicillin V) has become an official product of the British Pharmacopoeia 1958. The monograph states a dose range of 125 mg. to 250 mg. and for this reason British Drug Houses announce that Distaquaine V 120 mg. and 240 mg. tablets will in future be available to conform with B.P. requirements as 125 mg. and 250 mg. strengths. The 60 mg. tablets and the 2 Distaquaine V Elixirs will remain unchanged.

The D.Q.V. range will therefore consist of the following preparations:

Distaquaine V 60 mg. Tablets.
Distaquaine V 125 mg. Tablets.
Distaquaine V 250 mg. Tablets.
Distaquaine V Sulpha Tablets.
Distaquaine V Elixir.
Distaquaine V Elixir Forte.

* * *

The certificate and badge of the highest honour which the South African Red Cross Society can confer (an Honorary Life Membership) have been presented to Dr. A. Miller for outstanding services to the Red Cross cause over a period of more than 20 years. The presentation was made in Johannesburg on 6 June 1958 by Dr. Lewis S. Robertson, Chairman of the Executive Committee of the South African Red Cross Society.

Dr. A. Miller

Dr. Miller, who resides in Benoni, has been Chairman of the National Technical Training Committee of the Red Cross since 1943. He edited the revised edition of the Society's First-Aid manual.

He is the twelfth recipient of an Honorary Life Membership certificate.

CELONTIN FOR EPILEPTIC PATIENTS

Celontin was used satisfactorily to treat 41 patients with petit mal, minor motor seizures, psychomotor seizures and a few cases of grand mal, according to a recent report from the Mayo Clinic, Rochester, Minn.

Dr. Haddow M. Keith and Dr. Joseph G. Rushton, in a published report (Proceedings of the Staff Meetings of The Mayo Clinic, Vol. 33, No. 5, March, 1958) said, 'Celontin is an anticonvulsant drug, useful in both children and adults.'

Treatment of 41 patients, between 3½ and 61 years old, consisted of from 0.3 g. to 1.8 g. of Celontin daily. The usual dosage was 0.3 g. 3 or 4 times daily.

Results of the Celontin study were classified in 3 ways:

(1) *Complete Control.* Attacks ceased entirely after the administration of Celontin (minimal period, 2 months);

(2) *Improvement.* Attacks were reduced appreciably, either in number or in severity or both, and the patient desired and was able to continue the medication (minimal period, 2 months); and

(3) *Failure.* No appreciable improvement.

The Mayo physicians reported: '12% of our patients were completely controlled when Celontin was given alone or added to the existing form of treatment, 54% were definitely improved, side effects occurred in about 30% necessitating withdrawal of the drug in 3 cases (rash in one; nausea, dyspnea and restlessness in another, and drowsiness in the third).'

Dr. Keith and Dr. Rushton concluded: 'approximately 66% of our patients were benefited or completely controlled.'

* * *

ANAESTHETIST

With acknowledgments to *Mr. Bob Connolly* and *The Star.*

Westdene Products (Pty.) Ltd., Surgical Division, advise that there was an error in the new Welch Allyn Wall Transformer Unit which was advertised in this Journal on 31 May 1958. This unit connects to regular 200–220 v. AC (not 110–120 v. AC, as stated).

Enquiries should be made to: Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

* * *

NUTRITION SOCIETY OF GREAT BRITAIN

The Nutrition Society will hold a symposium on *Nutrition and Teeth* at the London Hospital Medical School, Turner Street, London, E.1, on Saturday, 4 October 1958.

The Nutrition Society will hold an *Open Scientific Meeting* in London for the presentation of original papers and demonstrations by members, and others introduced by them on Saturady, 13 December 1958. Titles of contributions, each with an abstract not exceeding 400 words or the equivalent in space (including title and references) should be sent to:

Miss D. F. Hollingsworth, c/o Ministry of Agriculture, Fisheries and Food, Great Westminster House, Horseferry Road, London, S.W.1, by 7 November.

The Scottish Group of the Nutrition Society will hold an *Open Scientific Meeting* in Dundee for the presentation of original papers and demonstrations by members, and others introduced by them on Saturday, 7 February 1959. Titles of contributions, each with an abstract not exceeding 400 words or the equivalent space (including title and references) should be sent to:

Dr. J. Davidson, The Rowett Research Institute, Bucksburn, Aberdeenshire, by 20 December 1958.

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EFFECTIVENESS OF ASIAN INFLUENZA VACCINE REPORT FROM UNIVERSITY OF KANSAS

Two University of Kansas physicians reported (Journal of the Kansas Medical Society, Vol. LIX, No. 33, page 111, 1958) on the effectiveness of vaccination with Asian virus (200 C.C.A. units) during the 1957 epidemic of Asian 'flu.

Dr. Robert A. Jordan and Dr. Tom D. Y. Chin conducted their study on a group of 1,291 employees and students at the University of Kansas Medical Center.

The study, divided into two groups (febrile and non-febrile) was difficult because of the uncertainty in diagnosing Asian 'flu. However, it appears likely that the majority of cases (studied) of acute respiratory illness with fever were caused by the Asian influenza virus.

When all respiratory illnesses, both febrile and non-febrile were totalled, the incidence was 133 cases in 640 vaccinated individuals; and, in 651 unvaccinated persons, the incidence was 364.

Thus, the total over-all effectiveness of the vaccine against all respiratory illnesses was estimated to be 63 per cent.

POLIOMYELITIS*

The second *Report* of the Expert Committee on Poliomyelitis, which met from 15 to 20 July 1957, was very well received by the Executive Board (WHO) and provoked a lengthy discussion. Part of the *Report* deals with the large-scale use of inactivated vaccines and with problems associated with their production. Recommendations are made concerning the organization of vaccination programmes and serological surveys, and a better utilization of regional laboratories collaborating with WHO is advocated. A considerable portion of the *Report* is devoted to the possibility of field trials with live poliomyelitis virus vaccine and the specifications of strains to be used. The Committee suggested a classification to avoid confusion of poliomyelitis with other diseases; and it recommended further research on inactivated and live virus vaccines, on combinations of other immunizing agents with poliomyelitis antigens, and on improved laboratory procedures.

Professor Pesonen (*Finland*) praised the *Report* and offered a number of comments. The epidemiological studies advocated by the Committee, though providing the best data for assessing actual needs, required a great deal of work, time and money. He reported a field study recently carried out in Finland and not yet published. The distribution of poliomyelitis antibodies in 5 communities had been found to be statistically the same in every age-group. He agreed that the term 'non-paralytic poliomyelitis' should not be used, particularly during epidemics, as many cases of aseptic meningitis and meningo-encephalitis might easily be so described. The *Report* gave a very valuable account of diseases resembling poliomyelitis and it was important to disseminate that knowledge among workers in the field. He also endorsed the Committee's recommendation that experiments with live attenuated vaccines should be continued, and said it would only be a matter of time before it would be possible to use them on a wider scale. Their administration was technically easier and would meet with less opposition from those who resisted inoculations in principle.

Dr. Metcalfe (*Australia*) said that since 1955 Salk vaccine had been distributed in Australia as rapidly as it could be produced. Up to the present time, every child of school age had received 3 injections and some states had already started a vaccination programme for adults up to 45 years of age. Last year there had been only 138 cases of poliomyelitis compared with over 1,000 cases in previous years. There had been no complaints of reactions and no case of poliomyelitis following injections.

In answer to questions from the Chairman, Sir John Charles (*United Kingdom*), it was stated that, although there was laboratory evidence that antibodies were produced following intracutaneous inoculation, there was no statistically accepted field evidence of protection comparable to that from the United Kingdom and United States field trials. The Committee had taken the view that the practical results were not yet good enough to allow any margin for reducing the dose by means of intracutaneous inoculation or the use of adjuvants.

* From *Chronicle of the World Health Organization*, Vol. 12, No. 3, March 1958, p. 86.

PREPARATIONS AND APPLIANCES

PROCTOSEDYL ROUSSEL

OINTMENT AND SUPPOSITORIES FOR THE TREATMENT OF HAEMORRHOIDS

Proctosedyl brings into contact with the lesions 4 active principles, with anti-inflammatory, antipruritic, antibacterial and trophic properties, in 2 complementary forms.

Ointment

Hydrocortisone	5 mg.	(0.5%).
Cinchocaine HCl	5 mg.	(0.5%).
Soframycin	10 mg.	(1.0%).
Aesculin	10 mg.	(1.0%).

Water-miscible vehicle *q.s.* to 1 g.

Suppositories

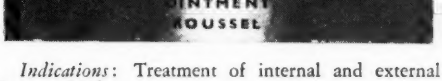
Hydrocortisone	5 mg.
Cinchocaine HCl	5 mg.
Soframycin	10 mg.
Aesculin	10 mg.

Cocoa butter *q.s.* for one suppository.

The Unforgettable Complaint: Haemorrhoids progress by successive acute episodes, during which the patient is constantly aware of his discomforting and distressing symptoms, which tend to interfere with his social activities and may lead to introspection and depression. The repeated haemorrhoidal attacks soon lead to haemorrhages and to chronic painful inflammatory lesions. In addition, haemorrhoids are very frequently complicated by pruritus ani, which may lead to peri-anal eczema.

Proctosedyl contains the necessary substances for rapid sedation of the pruritic and painful phenomena:

i. Hydrocortisone (Anti-inflammatory and antipruritic).
ii. Cinchocaine Hydrochloride (Local anaesthetic).
iii. Soframycin (Antibacterial).
iv. Aesculin (Increases capillary resistance and thus reinforces the action of hydrocortisone in controlling serous discharge).



Indications: Treatment of internal and external haemorrhoids.

Prophylaxis between attacks to prevent recurrences.

Complications of haemorrhoids: anal pruritus, peri-anal eczema, proctitis, acute fissure.

Patients awaiting haemorrhoidectomy.

Following haemorrhoidectomy.

Mode and Frequency of Application. Twofold Treatment: The two forms of *Proctosedyl* are complementary in their effects: the Ointment relieves the external symptoms while the Suppositories provide prolonged local action within the anal canal. (The use of the Ointment is indicated alone, however, during very painful crises accompanied by sphincteric spasm, which would prevent the introduction of a Suppository).

Proctosedyl Ointment: With the finger apply a small quantity of Ointment to the painful or pruritic area. Apply morning and evening and after each stool. (*Proctosedyl* Ointment does not stain fabrics).

Proctosedyl Suppositories: Insert a Suppository morning and evening and after each stool. It should be inserted as deeply as possible.

Packings: *Proctosedyl* Ointment 5 g. tubes.
Proctosedyl Suppositories Box of 6 Suppositories.

Manufactured by Roussel Laboratories Ltd., 847 Harrow Road, London.

South African Distributors: Fisons Chemicals (S.A.) (Pty.) Ltd., 226 Market Street, Johannesburg.

GONADYL (SERUM GONADOTROPHIN) TABLETS

FOR THE TREATMENT OF ACNE VULGARIS

Rationale. Serum gonadotrophin therapy stimulates the secretion of oestrogen, thus re-establishing the correct androgen : oestrogen ratio. A relative androgen excess is not, of course, the only aetiological factor, but there is considerable evidence that it does play an important role in the pathogenesis of acne vulgaris (one example being the appearance of acne during treatment with virilizing androgens).

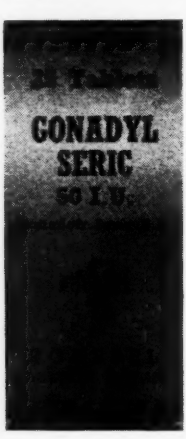
Advantages. With *Gonadyl* (serum gonadotrophin) Tablets, which avoid the inconvenience of implantation, the daily dosage is completely controllable throughout the course of treatment. Thus the possibility of excessive initial absorption of hormone, which might result in an exacerbation of the acne, is avoided.

No Side Effects. There are no side effects with *Gonadyl* Tablets.

In the female, the small amount of 100 I.U. (2 tablets) daily cannot have any deleterious effects on ovulation or menstruation. In fact, it is sometimes found that dysmenorrhoea or insufficient or excessive bleeding, if present, disappears with the treatment.

In the male, the small amount of serum gonadotrophin in *Gonadyl* Tablets could only stimulate, and definitely not inhibit, spermatogenesis.

Progress. During the treatment, improvements and exacerbations of the acne occur, final resolution or maximal improvement not taking place until the end of the course. The patient should be informed of this phenomenon so that the treatment may be continued without interruption and the course thus completed.



Dosage. The course of treatment lasts 12 weeks and requires a total of 100 tablets as follows:

2 *Gonadyl* Tablets daily for 4 weeks,

1 *Gonadyl* Tablet daily for 4 weeks.

1 *Gonadyl* Tablet on alternate days for 4 weeks.

(Please see note concerning storage).

It is essential for success that this dosage is not reduced in amount or duration.

MacKenna and Lipman Cohen used a dosage of 2 tablets daily for the whole period of 3 months, followed by slow reduction of dosage before discontinuing the treatment.

Results. Gonadyl Tablets are indicated only for acne vulgaris. (Acne rosacea is resistant to the treatment). The treatment is suitable for cases of all degrees of severity and for patients of both sexes. About 80–90% of cases are either cured or greatly improved (Aron-Brunetière 1954, 1955). R. M. B. MacKenna found this method of treatment to be worthy of further study (International Congress of Dermatology, Stockholm, 1957) and has reported that with it 60% of a series of cases showed great improvement and 25% improvement (*Lancet*, 1957, **2**, 600). E. Lipman Cohen has similarly obtained very good improvement in about 60% of patients (*Lancet*, 1957, **2**, 647).

Mode of Administration. Administration must be strictly by the *sublabial* or *sublingual* route, to allow full transmucosal absorption.

The best times for administration are in the evening on going to bed and in the morning just after breakfast. No drink or food should be taken for at least half an hour after the tablet has completely disappeared, so as to promote the transmucosal absorption of any hormone remaining in the mouth.

Packing. Bottles of 25 tablets each containing 50 I.U. of serum gondatrophin (follicle stimulating hormone, FSH).

Storage. Gonadyl Tablets may lose potency after prolonged exposure to air. The bottles are therefore filled and sealed under nitrogen. Their full potency cannot be guaranteed indefinitely after opening the bottle.

Manufactured by Roussel Laboratories Ltd., 847 Harrow Road, London.

South African Distributors: Fisons Chemicals (S.A.) (Pty.) Ltd., 226 Market Street, Johannesburg.

CELLULAR THERAPY: SICCACELL

Organ or cellular therapy is as old as the history of mankind itself. Küttner as far back as 1912 postulated intramuscular implantation of highly differentiated organs instead of surgical transplantation. In the same year, Carrel observed striking regenerative effects of embryonal heart muscle upon dying cultures of heart muscle. During the twenties Kurzahn, Hübner, Henschen and others made further researches on treatment by tissues and in 1931 Niehans put cellular therapy on a new and very extended basis.

The essential step could only be made about 5 years ago when intensive research on basic questions and clinical evaluation was made possible by the manufacture of lyophilized cell preparations (*Siccacell*). Cells from foetuses and young animals are preserved by lyophilization, an adequate method for preserving biologically active tissues.

The dried cells are checked for sterility. No infection has ever been reported after injection although over 1,000,000 preparations have been used so far. The incidence of allergic reactions was also small (about 6 per thousand). The reactions were of a mild nature.

The biological activity and therapeutic effect of dried cells can be proved by various methods. The preparations themselves contain enzymes (peroxidase and phosphatase) and various amino acids.

Although certain experimental studies indicate a special importance of various nucleic acid groups particularly to be found in mitochondria, experimental work with isolated cell elements, like mitochondria, and organ extracts gave therapeutic results

much inferior to those effected by whole cells. It appears that the active principle is bound to the whole cell.

Dried cells act by stimulating the corresponding organ or system of organs to functional regeneration. The question whether a genuine anatomical regeneration might be possible is not settled yet. According to its mode of action *Siccacell* therapy is only indicated where there is a sufficient rest of tissue susceptible to stimulation and reactivation. *Siccacell* therapy is not a substitution treatment.

Clinical trials on a wide scale have corroborated the experimentally observed stimulating action of cells. To our present knowledge the following groups of diseases may be regarded as acknowledged indications for *Siccacell*:

Atherosclerosis and circulatory disorders; endocrine insufficiency symptoms; climacteric conditions and presenile involution; underdevelopment in children; chronic diseases of individual organs, e.g. myocardial degeneration, liver disease and the nephrotic syndrome.

It will be possible in the near feature to assess the limits and possibilities of this therapy even more accurately. This much may be said though: dried cell therapy is innocuous and it is very effective in a high percentage of the cases.

Sole South African Agents: Newport Trading Corporation Ltd., 15 Sydenham Road, Fordsburg, Johannesburg.

REFERENCES

Griffel, A. (1957): Arch. Pediat., **74**, 325.
Knüchel, F. and Kuhn, W. (1955): Die Medizinische, 16 April.

(*See advertisement on p. xxv*)

PREPARATE EN TOESTELLE

PROCTOSEDYL ROUSSEL

SALF EN STEEKPILLE VIR DIE BEHANDELING VAN AAMBEIE

Proctosedyl bring 4 aktiewe beginsels met ontstekingswerende, jeukverdrywende, bakteriebestrydende en trofiese eienskappe in twee aanvullende vorms in regstreekse aanraking met die letsels.

Salf

Hidrokortisoon	5 mg. (0.5%).
Kinchokaïen-HCl	5 mg. (0.5%).
Soframycin	10 mg. (1.0%).
Aesculin	10 mg. (1.0%).

Draer wat in water oplosbaar is *q.s.* tot 1 g.

Steekpille

Hidrokortisoon	5 mg.
Kinchokaïen-HCl	5 mg.
Soframycin	10 mg.
Aesculin	10 mg.

Kakaobotter *q.s.* vir een steekpil.

Die Kwaal wat 'n Mens nie kan Vergeet nie. Die vordering van aambeie word deur agtereenvolgende akute episodes gekenmerk, en gedurende hierdie episodes is die pasiënt gedurig bewus van sy ongerieflike en ontstellende simptome wat nie alleen sy sosiale bedrywighede versteur nie, maar ook aanleiding tot selfondersoek en neerslagtigheid gee. Die herhaalde aambei-aanvalle loop eerlank uit op bloeding en chroniese, pynlike ontstekingsletstels. Daarbenewens word aambeie dikwels gekompliseer deur pruritus ani wat op sy beurt weer aanleiding tot peri-anusekseem kan gee.

Proctosedyl bevat die nodige middels vir die vinnige kalmering van die gejeuk en die pynlike verskynsels:

i. Hidrokortisoon (werk die ontsteking en die gejeuk teë).

ii. Kinchokaïenhidrochloried ('n plaaslike verdowingsmiddel).

iii. *Soframycin* ('n bakteriebestrydende middel).

iv. *Aesculin* (versterk haarvatweerstand en verhoog dus die hidrokortisoon se vermoë om weiagtige afskeiding te kontroleer).

Indikasies: Vir die behandeling van inwendige en uitwendige aambeie.

Profilakse tussen aanvalle om 'n herverskyning van die kwaal te voorkom.

Die komplikasies van aambeie; en pruritus ani, peri-anus-ekseem, nersdermontsteking, akute barste.

Pasiënte wat op aambei-uitsnyding wag.

Na uitsnyding van die aambeie.

Aanwendingsmetode en -Frekwensie. Dis 'n tweeledige behandeling. Die twee vorms van *Proctosedyl* het 'n aanvullende effek. Die salf verlig die uitwendige simptome, terwyl die steekpille 'n langdurige plaaslike effek binne in die anus-kanaal het. Tydens die baie pynlike krisisse wat met sulke kwaai ringspierkrampe gepaard gaan dat dit die insteek van die steekpil verhinder, is dit raadsaam om alleen die salf te gebruik.

Proctosedyl-Salf: Smeer 'n klein bietjie van die salf met die vinger aan die pynlike en jeukende dele. Wend soggens en saans en na iedere ontlasting aan. (Klerestowwe word nie deur *Proctosedyl*-salf bevlek nie).

Proctosedyl-Steekpille: Steek 'n steekpil soggens en saans en na iedere ontlasting in. Dit moet so diep moontlik ingesteek word.

Verpakking: *Proctosedyl*-Salf, buisies van 5 g.
Proctosedyl-Steekpille, dosies van 6.

Voorberei deur Roussel Laboratories Ltd., Harrowweg 847, Londen.

Suid-Afrikaanse Verspreiders: Fisons Chemicals (S.A.) (Pty.) Ltd., Markstraat 226, Johannesburg.

GONADYL (SERUM GONADOTROFIEN) -TABLETTE

VIR DIE BEHANDELING VAN GEWONE AKNEE

Verduideliking. Serum-gonadotrofien-terapie bevorder die afskeiding van estrogeen en herstel dus die korrekte androgeen-estrogeen-ewewig. 'n Betreklike oormaat aan androgeen is natuurlik nie die enigste etiologiese faktor nie, maar daar is aansien-

like bewyse dat dit 'n belangrike rol in die patogenese van gewone aknee speel. Een voorbeeld is die verskyning van aknee tydens behandeling met viriliserende androgene.

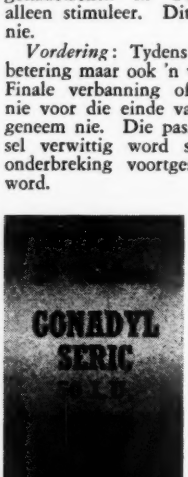
Voordele: Met *Gonadyl* (serum gonadotrofien) -tablette wat nie die ongerief van implanting meebring nie, is dit moontlik om die daaglikse dosis volkome te beheer solank die behandeling voortduur. Die moontlikheid van oormatige aanvanklike opneming van die hormoon wat bes moontlik op verergering van die aknee kan uitloop, word dus vermy.

Geen Newe-Effekte Nie: Daar is geen nêweeffekte met *Gonadyl*-tablette nie.

By die vrou kan die klein hoeveelheid van 100 I.E. (2 tablette) per dag geen nadelige effek op eieruitstoting of menstruasie hê nie. Inderdaad daar word soms gevind dat dismenorree of onvoldoende of oormatige bloeding, indien aanwesig, verdwyn as 'n vrou met hierdie middel behandel word.

By die man kan die klein hoeveelheid serum gonadotrofien in *Gonadyl-tablette* spermatogenese alleen stimuleer. Dit kan dit beslis nie onderdruk nie.

Vordering: Tydens behandeling vind daar 'n verbetering maar ook 'n veregering van die aknee plaas. Finale verbanning of maksimum-verbetering word nie voor die einde van die behandelingskuur waargeneem nie. Die pasiënt moet van hierdie verskynsel verwittig word sodat die behandeling sonder onderbreking voortgesit en die kuur voltooi kan word.



Dosis: Die behandeling duur 12 weke lank en vereis altesaam 100 tablette soos volg:

2 *Gonadyl*-tablette per dag gedurende 'n tydperk van 4 weke.

1 *Gonadyl*-tablet per dag gedurende 'n tydperk van 4 weke.

1 *Gonadyl*-tablet al om die ander dag gedurende 'n tydperk van 4 weke.

(Lees asseblief ook die aantekeninge oor die wyse waarop hierdie tablette gebêre moet word).

Vir die welslae van die behandeling is dit noodsaaklik dat bostaande dosisse nie verminder moet word wat hoeveelheid of tydsduur betref nie.

MacKenna en Lipman Cohen het 'n dosis van 2 tablette per dag gedurende die hele tydperk van 3 maande gebruik; daarna is die dosis stadig verminder vóór die staking van die behandeling.

Resultate. *Gonadyl*-tablette word aangedui slegs vir die behandeling van gewone aknee. (Acne rosacea sal nie op hierdie middel reageer nie). Die behandeling is geskik vir alle gevalle—van die ligste tot die ergste—en vir pasiënte van albei geslagte. In 80–90% van die gevalle vind daar 'n genesing of 'n aansienlike verbetering plaas (Aron-Brunetière, 1954, 1955). R. M. B. MacKenna het bevind dat hierdie behandelingsmetode verdere studie regverdig (Internasionale Kongres oor Dermatologie, Stockholm, 1957). By 'n reeks pasiënte wat op hierdie manier behandel is, rapporteer hy, was daar aansienlike verbetering by 60%, en verbetering by 'n verdere 25% (*Lancet*, 1957, **2**, 600). E. Lipman Cohen het op dieselfde manier daarin geslaag om baie goeie beterskap teweeg te bring by ongeveer 60% van sy pasiënte (*Lancet*, 1957, **2**, 647).

Toedieningsmetode: Toediening moet alleen langs die *onderlip*- of *ondertong*-roete geskied om volledige transslymvlies-absorpsie in die hand te werk.

Die beste tye om die tablette te neem, is net voordat die pasiënt saans gaan slaap en in die oggend net nadat hy sy ontbyt geniet het. Om die transslymvlies-absorpsie van enige hormone wat in die mond agtergebly het te bevorder, moet geen vloeistowwe of voedsel geneem word gedurende die eerste halfuur volgende op die algehele verdwyning van dit tablet nie.

Verpakking: Bottels van 25 tablette, elk bevattende 50 I.E. serum gonadotrofien (follokel-stimulerende hormoon, FSH).

Wegbêre. *Gonadyl*-tablette kan hul krag verloor as hulle lank aan lug blootgestel word. Die bottels word derhalwe gevul en verseël onder stikstof. Hul volle kragtigheid kan nie 'n onbepaalde tyd lank ná die oopmaak van die bottel gewaarborg word nie.

Voorberei deur Roussel Laboratories Ltd., Harrowweg 847, Londen.

Suid-Afrikaanse Verspreiders: Fisons Chemicals (S.A.) (Pty.) Ltd., Markstraat 226, Johannesburg.

SELLULÊRE TERAPIE: SICCACELL

Orgaan- of sellulêre terapie is so oud soos die geskiedenis van die mensdom self. Binnespierse inplanting van hoogs gedifferensieerde organe in plaas van chirurgiese oorplanting is reeds sover terug as 1912 deur Küttner gepostuleer. Die opvallende regenerasie-effek van embrionale hartspier op sterwende hartspierkwekinge is gedurende dieselfde jaar deur Carrel waargeneem. In die twintigier jare van hierdie eeu het Kurzahn, Hübner, Henschen en andere verdere navorsingswerk op die gebied van behandeling met weefsels gedoen, en in 1931 het Niehans sellulêre terapie op 'n nuwe en aansienlik uitgebreide grondslag gestel.

Die noodsaaklike stap kon egter eers ongeveer 5 jaar gelede gedoen word toe intensiewe navorsing in verband met basiese vraagstukke en kliniese evaluasie moontlik gemaak is deur die vervaardiging van geliofiliseerde selfpreparate (*Siccacell*). Selle afkomstig van fetusse en jong diere word gepreserveer deur liofilisasie, 'n doeltreffende metode vir die bewaring van biologies aktiewe weefsels.

Die droë selle word gekontroleer vir steriliteit. Geen infeksie na 'n inspuiting is gerapporteer nie, hoewel meer as 1,000,000 preparate tot dusver gebruik is. Die voorkoms van allergiese reaksies was ook klein (ongeveer 6 per duisend), en die reaksies was van 'n geringe aard.

Die biologiese aktiwiteit en terapeutiese effek van droë selle kan met behulp van verskillende metodes bewys word. Die preparate self bevat ensieme (peroksidase en fosfatase) en verskillende aminosure.

Hoewel sekere proefondervindelike studies die spesiale belangrikheid aangedui het van verskillende nukleïensuurgroepe wat in mitokondrië aangetref word, het eksperimentele werk met geïsoleerde selelemente, soos mitokondrië, en orgaanuittreksels terapeutiese resultate opgelewer wat veel swakker was as dié waarop heel selle ingewerk het. Dit skyn asof die aktiewe beginsel aan die heel sel verbonde is.

Droë selle werk deur die ooreenstemmende orgaan of orgaanstelsel tot funksionele regenerasie te stimuleer. Die vraag of egte anatomiese regenerasie moontlik is, is nog nie uitgemaak nie. Volgens sy werkingsmetode word *Siccacell*-terapie alleen aangedui wanneer daar 'n voldoende oorskot is van weefsel wat vir stimulasie en reaktivering vatbaar is. *Siccacell*-terapie is nie 'n aanvullingsbehandeling nie.

Kliniese proefnemings in 'n uitgestrekte gebied het die eksperimenteel waargenome stimulasie-effek van selle bevestig. Sover ons op die oomblik weet, kan die volgende groepe siektes as erkende indikasies vir *Siccacell* beskou word:

Aterosklerose en bloedsomloopkwale; simptome van 'n endokriengebrek; klimakteriumtoestande en pre-seniele involusie; gebrekkige ontwikkeling by kinders; chroniese kwale van individuele organe, bv. hartspierdegenerasie, lewerkwaal en die nefrotiese sindroom.

In die naaste toekoms sal dit moontlik wees om die perke en die moontlikhede van hierdie terapie selfs akkurater vas te stel. Intussen kan dit egter gesê word: droë-sel-terapie is onskadelik, en in 'n hoë persentasie van gevalle is dit besonder doeltreffend.

Alleenagente in Suid-Afrika: Newport Trading Corporation Ltd., Sydenhamweg 15, Fordsburg, Johannesburg.

VERWYSINGS

Griffel, A. (1957): Arch. Pediat., **74**, 325.
Knüchel, F. en Kuhn, W. (1955): Die Medizinische, 16 April.

(*Lees ook die advertensie op bl. xxv*)

REVIEWS OF BOOKS

LEPTOSPIROSIS

Leptospirosis in Man and Animals. By J. M. Alston, M.D., F.R.C.P. (Edin.), and J. C. Broom, *O.B.E.*, M.D. 1958. (Pp. 344 + Index. With Figs. 40s.). Edinburgh and London: E. & S. Livingstone Ltd.

This book presents all the data in connexion with leptospirosis in Man and animals and will serve as a work of reference to the bacteriologist, epidemiologist, clinical pathologist, physician and public health officer as well as to those concerned with the medico-legal aspect of the problem. Each sero-type is described both from the bacteriological and clinical aspects and much that is new to the practising bacteriologist and clinical pathologist is presented in detail. Of especial importance to the clinician are the descriptions and case histories of the various sero-types of the Leptospires, their clinical diagnosis and laboratory diagnostic methods. All aspects of the occurrence of the infection in America, Europe, Asia, Australia and New Zealand are described and the occupations at risk.

The disease occurs in the rat-infested sugar-cane fields of Queensland, in wet rice growing areas, in mines, in general farm work including grain harvesting, in veterinary work, pig breeding, cattle raising and dog kennel work, in sewer workers and in food handling—slaughtering, meat packing, fish cleaning and poultry dressing. This extensive list of occupations and the mild form in which the disease is often manifested suggests that in South Africa its existence might not have been recognized. In this regard the recent report in Johannesburg of several cases of *L. canicola* meningitis is of especial importance, and the publication of this book is welcomed enthusiastically as it could stimulate both clinicians and laboratory workers to detect cases of leptospirosis, especially in the recently established rice growing areas in the Union and the sugar cane regions in Natal.

The importance of the infection in regard to the Workmen's Compensation Act is obvious. The book is recommended to medical officers of health, physicians, bacteriologists and clinical pathologists as an essential addition to their library. The list of references and the bibliography are both very extensive.

COLPOMICROSCOPY

Atlas of Colpomicroscopy. By Prof. T. Antoine and Dr. V. Grünberger. 1956. (Pp. 224 + viii. Illustrations in full colour. D.M. 64.) Stuttgart: Georg Thieme Verlag.

The accessibility of the cervix to direct observation has stimulated gynaecologists and pathologists to devise ingenious techniques intended to provide early diagnosis of malignancy or pre-malignancy. In Anglo-American circles, largely under the pioneering influence of Papanicolaou and Traut, emphasis has been on sampling of the desquamated cell population in the cervix and vaginal fornices with a search for the occasional malignant cell which may have been shed.

The gamble inherent in such methods is obvious, but equally obvious is the value of a diagnostic technique which might render unnecessary the performance of formal cervical biopsy for histological examination. The difficulty, and the inherent weakness, of the latter method is the selection of a suitable area of the cervix for excision, a difficulty which some workers have solved to some extent by the rather elaborate procedure of conization of the cervix with complete histological study of the entire critical zone of the cervix.

On the Continent, however, particularly in Austrian and German circles, the approach has been to devise ever more elegant methods of identifying and removing suspicious zones for study. The first and simplest method was Schiller's test with Lugol's iodine. But more than 30 years ago Hinselmann devised his colposcope, which enabled direct observation of the cervix magnified 10–20 times.

Much later Antoine and Gruneberger, of the famous Frauenklinik of the University of Vienna, developed from Hinselmann's colposcope a device enabling them to do what was virtually a histological examination of the cervix in a living patient. They named this the *colpomicroscope*. It consists of a tube which is introduced into the vagina and fixed in position. To it are connected the optical and lighting components, and examination of the unstained cervix can reveal the vascular pattern, the capillaries and the erythrocytes flowing through them. On staining with a modified haematoxylin solution, a picture similar to the ordinary histological picture is revealed.

This book is an *Atlas* covering the whole field of colpomicroscopy, illustrating by means of 176 black-and-white photographs (a camera is easily incorporated into the instrument) the various normal variations and pathological conditions of the cervix correlated with histological sections. The photographs are generally of a high standard and some are quite remarkable, particularly those comparing the vascular patterns in normal and malignant cervices. The latter constitutes an unusual approach to gynaecological pathology reminiscent of the angiographic studies of bone tumours currently attracting attention.

It is difficult to perceive the need for the indifferent plates demonstrating cyclical changes in the vaginal mucosa (which occupy pp. 195–207) unless it be to demonstrate the versatility of the instrument which can, of course, be applied to any other epithelial surface which can be reached by the tube.

The *Introduction* and the *Discussion* are in German, and only the technique appears in English, but all the photographs have legends in German, English and Spanish, the last a significant reminder of the growth of South American science in recent times.

Among those familiar with this work (and knowledge of it West of the English Channel was delayed by the last war) this publication is long-awaited and welcome; but it must not be assumed that this ingenious instrument is the final answer to cervical pathological diagnosis. Much of the reason for the great interest in the cervix, and the innumerable publications dealing with it, is the frequency with which carcinoma-*in-situ* is diagnosed. That this is in fact an entity has been disputed for a considerable time. Its role as a precursor to invasive carcinoma has been suggested, since it is frequently found near areas of invasion or on the hypothesis that, left untreated, progression to invasiveness is inevitable. It is too often forgotten by clinicians (whose faith in the pathologist, although flattering, may be dangerous) that the evaluation of biopsies is limited solely to the condition in the small piece of tissue removed. It is all too easy to miss adjacent invasiveness. Consequently any technique which will improve the value of *sampling* must be welcomed as an aid to accuracy.

It is doubtful, however, whether it is possible to differentiate atypical hyperplasia, or dysplasia, from carcinoma-*in-situ* by this means. We all know how difficult it can be in a formal section, and this only serves to emphasize the value of both colposcope and colpomicroscope in pin-pointing suspicious lesions.

Statistically it was found that a combination of cytological, histological and colpomicroscopic study yielded the best chance of diagnosing pre-invasive cancer. With the simple colposcope alone (with much lower magnification) by merely finding abnormal areas and biopsying them, Professor Navratel in Graz, found many cancers of the cervix in individuals with negative vaginal cytology. That there is a strong case for the instrument is beyond doubt. But it is extremely doubtful whether the technique or the interpretations can be learnt from this or any other textbook. Ideally one should work at the Continental clinics where the method is in current use. It is a valuable addition to our armamentarium of investigation, but does not replace in any way conventional curettage and histological study. It enables the observer to aim his sharp pointed curette at the very lesion of the cervix, and obtain minute fragments of tissue that obviously show cancer, as well as permitting detailed study of the entire cervix at high magnification.

Professor Antoine and Dr. Gruneberger have produced an outstanding monograph which really makes this work available for the first time in English (or partly so). It is noteworthy that in a bibliography of over 30 references in it and related fields, not one is in the English language, and it is to be hoped that the workers in this country (this reviewer has heard that several instruments are already in use here) will be stimulated by this brilliant contribution to study its utilization in an area where early diagnosis of malignant disease leads to the most gratifying results in reducing both morbidity and mortality.

ACUTE POISONING

Handbook of Treatment of Acute Poisoning. By E. H. Bensley, *M.B.E.*, B.A., M.D., F.A.C.P. and G. E. Joron, B.A., M.D., C.M., F.A.C.P. 2nd ed. 1958. (Pp. 206 + Index. 15s.). Edinburgh and London: E. & S. Livingstone Ltd.

One of the serious gaps in the instruction of the undergraduate medical student is the treatment of acute poisoning—an emergency which brooks no delay in its management. This hiatus in his training means that the provisionally registered medical practitioner (the intern) starts off his professional career in a hospital with a severe handicap. The need for a handbook specifically confined to the treatment of acute poisoning is therefore extremely welcome and should have very considerable appeal to students as well as the fledgling doctor.

This is the second edition of Bensley and Joron's *Handbook*. The authors are associated with the Montreal General Hospital, and the Departments of Metabolism, Toxicology and Medicine. They are therefore particularly well placed to deal with and to teach the hazards of acute poisoning.

In the first section of the volume, the authors emphasize the importance of certain basic principles which must be mastered before the specific types of acute poisoning (described in the second section) can be appreciated to the best advantage. Prevention of further exposure to the poison, the maintenance of a clear airway, control of respiratory depression, shock, convulsions, infection and removal and inactivation of the poison, are dealt with succinctly in the first few pages of the manual. Adequate First Aid and early attention to the emergency may obviously not only be life-saving, but also do much to prevent complications.

The descriptions of the different types of acute poisoning and their treatment cover most of the emergencies the medical practitioner is likely to meet. The authors, very understandably, do not include the whole gamut of industrial poisons, as these fall within a rather special category, and come into the province of practitioners of industrial medicine.

The contents of an emergency poison kit are listed in an *Appendix*, and adequate references are given to works dealing specifically with the management of poisoning in childhood.

This *Handbook* should have an appeal not only to the medical profession, but also to such organizations as the Red Cross and St. John, whose courses of instruction in First Aid include the treatment of acute poisoning.